DISSERTATION ON

ECHOCARDIOGRAPHIC CHANGES ASSOCIATED WITH SYSTEMIC HYPERTENSION IN HYPERTENSIVE PATIENTS IN GOVERNMENT ROYAPETTAH HOSPITAL, CHENNAI – AN OBSERVATIONAL STUDY

Submitted to

The Tamil Nadu Dr. M.G.R. Medical University

In partial fulfillment of regulations for the award of the degree of

M.D. GENERAL MEDICINE

BRANCH – I

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KILPAUK MEDICAL COLLEGE

CHENNAI – 10

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CHENNAI

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"ECHOCARDIOGRAPHIC CHANGES ASSOCIATED WITH SYSTEMIC HYPERTENSION IN HYPERTENSIVE PATIENTS IN GOVERNMENT ROYAPETTAH HOSPITAL, CHENNAI – AN OBSERVATIONAL STUDY"** is a bonafide work done by **Dr.SRIDHAR. S.**, post graduate student, Department of General Medicine, Kilpauk Medical College, Chennai-10, under our guidance and supervision in partial fulfillment of the rules and regulations of **The Tamilnadu Dr.M.G.R.Medical University** for the award of **M.D.Degree Branch I, (General Medicine)** during the Academic period from May 2010 to March 2013.

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I, solemnly declare that the dissertation entitled "**ECHOCARDIOGRAPHIC CHANGES ASSOCIATED WITH SYSTEMIC HYPERTENSION IN HYPERTENSIVE PATIENTS IN GOVERNMENT ROYAPETTAH HOSPITAL, CHENNAI – AN OBSERVATIONAL STUDY"** is done by me at Kilpauk Medical College, Chennai – 10 during May 2010 to March 2013 under the guidance and supervision of **Prof.Dr.K.T. JAYAKUMAR, M.D.,** to be submitted to **The Tamilnadu Dr.M.G.R.Medical University** towards the partial fulfillment of requirements for the award of **M.D. DEGREE IN GENERAL MEDICINE BRANCH – I.**

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ABSTRACT

INRODUCTION

Hypertension is a major non-communicable disease which has major impact on morbity and mortality. Early detection of end organ damage may reduce the morbidity substantially

AIM OF THE STUDY

To study the prevalence of echocardiographic changes in hypertensive patients and to investigate correlation of echocardiographic changes with age, sex, duration and control of hypertension, tobacco use and body mass index.

MATERIALS AND METHODS

Patients with hypertension were recruited from hypertension clinic. A total of 100 patients who met inclusion and exclusion criteria were inducted into the study. Echocardiographic examination was performed to assess cardiac structural changes and cardiac function. The results were analyzed statistically

RESULTS AND OBSERVATIONS

In our study population, 64% had echocardiographic abnormality of left ventricle, either abnormal LV mass or LV Diastolic dysfunction out of which 27% had both. 50% of the sample group had LV Diastolic dysfunction while 41% had abnormal LV mass. Overall 63% of those with abnormal LV mass

were females. Even when the duration of hypertension is less than 5 years, 37% hypertensives had abnormal LV mass. 90% of those who had abnormal LV mass were tobacco users. Among obese hypertensives, 45% had abnormal LV mass and 57% had LV diastolic dysfunction. It is most important to note that 74% of patients has inadequate control of BP (SBP>140 mmHg and/or DBP > 90mmHg as per JNC7 guidelines)

CONCLUSION

In this study, half of asymptomatic hypertensives had evidence of LV diastolic dysfunction and even a short duration of hypertension results in abnormal LV mass and three-fourths of hypertensives have inadequate control of BP and two thirds of hypertensives were obese. This emphasizes importance of need for life style modifications and earlier achievement of target BP and implies the importance of screening of asymptomatic hypertensives for LV diastolic dysfunction.

CONTENTS

INTRODUCTION

1. INTRODUCTION

Systemic hypertension is a most common risk factor for ischaemic heart disease, peripheral vascular disease, aortic dissection, stroke, particularly heart failure, atrial fibrillation and also easily identified and reversible. Hypertension is a major global health problem affecting the public and a major cause of death globally. Since it is of asymptomatic nature, diagnosis is often delayed.

To treat systemic hypertension effectively, continued and frequent follow-up is necessary which is not that common among men and low income groups. Most of the time, a single disease causing mechanism could not be identified and treatment is empiric. Insufficient time for education, Medication burden, drug costs, and side effects of medication leads to non-adherence. Physicians also often hesitate to start and intensify anti-hypertensive medication.

Due to above mentioned reasons, Blood Pressure (BP) is controlled to a value less than 140/90 mm Hg in not more than one third of hypertensives, even in developed countries where most advance health care systems are available. Even when BP meets current standards, less than one in three are protected from complications. There is a probability of 90% chance of development of hypertension in a middle aged or elderly individual in their life time. Hence, assessment of risk of cardiovascular disease should be included in the

evaluation of both individuals at risk of developing systemic hypertension and hypertensive patients.

AIMS AND OBJECTIVES

2. AIM OF STUDY

- To study the prevalence of echocardiographic changes associated with systemic hypertension among hypertensive patients attending hypertension clinic at Govt. Royapettah hospital, Chennai

- To correlate the echocardiographic changes observed with age, sex, duration of hypertension, control of hypertension, smoking, BMI

REVIEW OF LITERATURE

3. REVIEW OF LITERATURE

Hypertension is a leading cause of global disease burden and an increasingly important public health problem. In the year 2001, approximately 13–15% of the total deaths and 92 million DALY worldwide were attributable to high blood pressure. The risk of cardiovascular disease is doubled with hypertension along with ischemic and hemorrhagic stroke, peripheral arterial disease and chronic kidney disease. It has been found a large percentage of hypertensive population were either inadequately treated or untreated. Observational studies involving greater than 1 million individuals have shown that there is a linear progression in mortality from CAD and stroke from BP levels as low as SBP 115 mmHg and DBP 75 mmHg upward.[63] As per Framingham Heart Study , there is a two fold increase of cardiovascular disease risk (relative risk) for BP values between 130–139/85–89 mmHg.[64]

3.1 EPIDEMIOLOGY

Affecting 1 billion people worldwide, hypertension is one of the common, easily identifiable conditions which affects about one billion people all over the world and is also reversible with proper follow-up and treatment. Hypertension mat lead to stroke, heart failure, myocardial infarction, arrhythmias, peripheral vascular disease and aortic dissection. The global

burden of hypertension is rising and projected to affect 1.5 billion persons—one third of the world's population—by the year 2025 due to aging population and increasing incidence of obesity.Currently, high blood pressure (BP) causes about 54% of stroke and 47% of ischemic heart disease worldwide.[1]

Half of this disease burden is in people with hypertension; the other half is in people with lesser degrees of high BP (prehypertension). Thus, high BP is the leading cause of mortality globally and leading public health problem in the world.

The asymptomatic nature of the condition delays diagnosis. Effective treatment requires continuity of care by a knowledgeable physician and frequent medical checkups, which are less common in men and low-income minorities.[2]

Even among patients whose hypertension control meets current standards, fewer than one in three is protected from end organ damage like heart failure, myocardial infarction or stoke.

Blood pressure levels, the rate of age-related increases in blood pressure, and the prevalence of hypertension vary among populations. Hypertension is not prevalent in a small number of individuals living in primitive, culturally isolated societies. In industrialized societies, blood pressure consistently increases in the first 10 -20 years of life. In younger age, blood pressure is associated with growth and maturation. Blood pressure "tracks" over time in

children and between adolescence and young adulthood. In the United States, blood pressure is higher in men than in women in early adulthood. Among older individuals, women had steeper increase in age-related rate of rise of blood pressure. Consequently, among individuals age 60 and older, systolic blood pressures of women are higher than those of men. Diastolic blood pressure increases progressively until ~55 years among adults, after which it tends to decrease. The consequence is a widening of pulse pressure (the difference between systolic and diastolic blood pressure) beyond age 60. There ia a 90% probability of developing systemic hypertension in the life time of a middle-aged or elderly person.

The incidence of hypertension in increasing with age there is a 65.4% prevalence among persons aged > 60 years. Recent evidence suggests that the prevalence of hypertension may be increasing, possibly as a consequence of increasing obesity.

Racial and regional variations in blood pressure prevalence may be contributed by b genetic and environmental factors. Weight gain and obesity are found to be strong, independent risk factors in hypertension. Sedentary life style, alcoholism, work related stress also may contribute to hypertension. Incidence of hypertension is 3.8 times more frequent in persons with positive family history of systemic hypertension.

As per 2010 Non-communicable disease report for india published by WHO 32.5% of Indians had raised blood pressure of prevalence among males (33.2%) and females (31.7%) was almost equal.[3]

3.2 DEFINING HYPERTENSION

Clinically, hypertension may be defined as that level of blood pressure at which the institution of therapy reduces blood pressure–related morbidity and mortality. Current clinical criteria for defining hypertension generally are based on the average of two or more seated blood pressure readings during each of two or more outpatient visits.

JNC 7 recommends Blood pressure criteria for defining hypertension was recommended by JNC7[4] . Patients were classified as having normal blood pressure, prehypertension, hypertension (stages I and II). The JNC 7 classification does not take in to account the target organ damage or presence or absence of risk factors in making recommendations for treatment, if both or either be present. As per JNC7 recommendation all persons with stage 1 and stage 2 systemic hypertension should be treated. Blood pressure <140/90 mmHg should be taken as the goal for persons with systemic hypertension and no other compelling indications. Compelling indications involve high-risk conditions that can be direct sequelae of hypertension (HF, IHD, chronic kidney disease, recurrent stroke) or commonly associated with hypertension (diabetes, high coronary disease risk). Therapeutic decisions in such individuals should be

directed at both the compelling indication and BP lowering. The goal for persons having no compelling indications and prehypertension is to control BP to normal blood pressure levels with lifestyle modifications and prevent the progressive rise in BP.

Table 3. Classification of blood pressure for adults

Blood Pressure Classification	SBP mmHg	DBP mmHg
Normal	<120	and <80
Prehypertension	120–139	or 80–89
Stage 1 Hypertension	140–159	or 90–99
Stage 2 Hypertension	≥160	or ≥100

SBP, systolic blood pressure; DBP, diastolic blood pressure

Fig 3.2.1 BLOOD PRESSURE IN ADULTS (AS PER JNC7 GUIDELINE)[4]

Clinic measured blood pressure levels are higher than Home measured blood pressure and average 24-h ambulatory measured blood pressure measurements are generally lower than clinic blood pressures. Increasing evidence suggests that home blood pressures, including 24-h blood pressure recordings, more reliably predict target organ damage than do office blood pressures. Blood pressure was found to be high in early morning of the, soon after waking. Nighttime measured blood pressures aer 10–20% lower than blood

pressures measured during day time, and an attenuated nighttime blood pressure "dip" is associated with increased cardiovascular disease risk. Average awake measured BP ≥135/85 mmHg and asleep measured blood pressure ≥120/75 mmHg approximate clinic measured blood pressure of 140/90 mmHg.

Approximately 15–20% of patients with stage 1 hypertension based on office blood pressures have average ambulatory readings <135/85 mmHg. This phenomenon, so-called white coat hypertension, also may be associated with an increased risk of end organ damage although to a lesser extent than in individuals with elevated office and ambulatory readings and is also associated with increased chance of sustained systemic hypertension.

The minimal laboratory testing required for the initial evaluation of hypertension includes determination of blood electrolyte values, fasting glucose concentration, and serum creatinine level, glomerular filtration rate, fasting lipid panel; hematocrit; spot urinalysis, including urine albumin-to-creatinine ratio; and resting 12-lead electrocardiogram.

3.3 CAUSES OF HYPERTENSION

Approximately 80–95% of hypertensive patients are diagnosed as having “primary” hypertension (also referred to as essential or idiopathic hypertension). In the rest 20% hypertensive patients, a specific secondary cause can be

identified. In addition to below mentioned causes, there is also a entity called mendelian form of hypertension.

Table 247–3. Secondary Causes of Systolic and Diastolic Hypertension	
Renal	Parenchymal diseases, renal cysts (including polycystic kidney disease), renal tumors (including renin-secreting tumors), obstructive uropathy
Renovascular	Arteriosclerotic, fibromuscular dysplasia
Adrenal	Primary aldosteronism, Cushing's syndrome, 17α-hydroxylase deficiency, 11β-hydroxylase deficiency, 11-hydroxysteroid dehydrogenase deficiency (licorice), pheochromocytoma
Aortic coarctation	
Obstructive sleep apnea	
Preeclampsia/eclampsia	
Neurogenic	Psychogenic, diencephalic syndrome, familial dysautonomia, polyneuritis (acute porphyria, lead poisoning), acute increased intracranial pressure, acute spinal cord section
Miscellaneous endocrine	Hypothyroidism, hyperthyroidism, hypercalcemia, acromegaly
Medications	High-dose estrogens, adrenal steroids, decongestants, appetite suppressants, cyclosporine, tricyclic antidepressants, monamine oxidase inhibitors, erythropoietin, nonsteroidal anti-inflammatory agents, cocaine

Fig 3.3.1 SECONDARY CAUSES OF HYPERTENSION [5]

3.4 HYPERTENSION AND CARDIOVASCULAR RISK

Heart disease in systemic hypertensive is due to anatomical and functional changes leading to left ventricular hypertrophy, Ischemic heart disease, heart failure, arrhythmias and Microvascular disease. Most common cause of mortality is Hypertension is heart disease.

Hypertension is a major risk factor not only for CAD but also for LVH and heart failure. Hypertension may contribute to CAD more than is commonly realized because hypertensives have more silent ischemia and unrecognized myocardial infarctions, and patients with acute myocardial infarction often have preexisting hypertension that evaded detection or treatment. Assessment of BP is inaccurate during an acute coronary syndrome because of pain-induced BP rise or dysautonomia or pump failure decreasing BP. Preexisting hypertension increases the case-fatality rate associated with an acute myocardial infarction and risk of hemorrhagic stroke is increased during thrombolytic therapy, especially when systolic BP exceeds 175 mm Hg. Cardiovascular risk also increases dramatically with hypertensive end organ damage.

TABLE 45-2 -- Risks Influencing Prognosis in Patients with Hypertension

Risk Factors for Cardiovascular Disease
Systolic and diastolic BP levels
Levels of pulse pressure (in the elderly)
Age: men >55 years; women >65 years
Smoking
Dyslipidemia (LDL-C >115 mg/dL)
Impaired fasting glucose (102-125 mg/dL) or abnormal glucose tolerance test result
Family history of premature cardiovascular disease
Abdominal obesity
Diabetes mellitus
Subclinical Target Organ Damage
Left ventricular hypertrophy
Carotid wall thickening or plaque
Low estimated glomerular filtration rate ≤60 mL/min/1.73 m^2
Microalbuminuria
Ankle-brachial BP index <0.9
Established Target Organ Damage
Cerebrovascular disease: ischemic stroke, cerebral hemorrhage, transient ischemic attack
Heart disease: myocardial infarction, angina, coronary revascularization, heart failure
Renal disease: diabetic nephropathy, renal impairment
Peripheral arterial disease

FIG 3.4.1RISK FACTORS INFLUENCING PROGNOSIS IN HYPERTENSION[6]

There is doubling of cardiovascular disease risk for every 20-mmHg increase in SBP and 10-mmHg increase in DBP. [7] Persons ranging from 40 to 89 years of age at increased risk. Data derived from Framingham Heart Study have shown that BP between 130–139/85–89 mmHg is associated with greater than twofold increased risk of cardiovascular disease (CVD) when compared with persons with BP levels lower than 120/80 mmHg.[8] Greater the BP, higher

the risk of myocardial infarction, heart failure, chronic kidney disease and stroke.[9]

Framingham CHD risk score calculated from tables published [10] assists physicians and hypertension patients in follow up of treatment.

3.5 HEART FAILURE AND HYPERTENSION

Heart failure caused most deaths from hypertension before the advent of effective drug therapy for hypertension in the late 1950s. Better management has substantially reduced hypertension-related deaths from heart failure and significantly delayed its onset, but hypertension remains the most common cause of heart failure with preserved systolic function. In addition, hypertension indirectly leads to systolic heart failure as a major risk factor for myocardial infarction. CHF may be related to Left Ventricular (LV) systolic dysfunction, LV diastolic dysfunction, or both.

In individuals with HF symptoms, 40-50% may have preserved LV systolic function. They have a high chance of having systemic hypertension, Left Ventricular Hypertrophy, isolated diastolic dysfunction. They are also more likely to be females. [11] [12] Progression to LV dysfunction can be greatly reduced by treatment with ACE Inhibitors, Beta blockers and diuretics. Heart failure is preceded by systemic hypertension in 90%. Risk of heart failure is also increased 2-3 fold in patients with hypertension. Hypertension in elderly

individuals is especially important. Heart failure incidence has remained unchanged in males and in females, HF has declined over past 50 years.[13] But, hospitalization due to HF have doubled in the past twenty years [14] probably secondary to improvement in therapeutic methods which result in increased life expectancy. With increasing proportion of aging population HF may become more prevalent in near future. probably become even more prevalent in the future as our population ages. Reducing SBP is uniformly beneficial, even though BP targets for Heart Failure have not been established firmly.

3.6 HEART FAILURE WITH PRESERVED EJECTION FRACTION (HFnlEF) [Diastolic Heart Failure]

Diastolic Heart Failure is said to be present when the ejection fraction at rest is normal or near normal. In this condition, heart is either normal or small in size and heart failure features are present. Left ventricular (LV) hypertrophy will be often present and filling of heart is impaired due to altering of LV stiffness. There will also be other evidence of LV diastolic Dysfunction. Conditions such as Severe Hypertension or Valvular heart like mitral regurgitation may be present. Diastolic heart failure may coexist with Systolic Heart Failure, especially during exercise

"Heart failure with preserved systolic function" is a heterogeneous group including noncompliant and stiff left ventricle dysfunction (isolated diastolic

heart failure), dysfunction of cardiac valves, cardiac arrhythmias, RV dysfunction, pericardial disease. It is thus a diagnosis of exclusion, and such a nonspecific approach can lead to many patients being given a diagnosis of heart failure incorrectly. Much controversy has arisen around the best noninvasive methods of identifying heart failure caused by isolated diastolic dysfunction, with European guidelines suggesting that to make this diagnosis, there should be Heart failure symptoms and signs, abnormal LV relaxation, normal (or only mildly abnormal) LV systolic function and filling, diastolic stiffness or distensibility.[15]

Such a definition has proven difficult to use in clinical practice. Vasan and colleagues have shied away from this strict definition and have suggested dividing the diagnosis of diastolic heart failure by the likely probability of the diagnosis being correct (definite, probable, or possible). Individuals are categorized depending on the absence and presence Heart Failure, with evidence of normal systolic function (EF >50%) during a heart failure event, and evidence of diastolic dysfunction chiefly from echocardiography. This approach was endorsed by the ACC/AHA. [16]

Heart failure with preserved systolic function was not considered to be common in many population based studies, but recent reports from Olmsted County (Rochester Epidemiology Project) and a small nested case-control study from the Framingham Heart Study in North America suggest this can be as

common as systolic dysfunction, particularly if the diagnosis of heart failure is based on accepting the clinician's opinion or using the Framingham criteria and relying on echocardiography at any point during a hospitalization period.[17] [18] [19] [20][21] Most of the studies suggest that the probability of the systolic function of the left ventricle being preserved in a patient with heart failure is higher in the elderly, women, and the obese.[22]

3.6.1 PROGNOSIS OF HEART FAILURE

Factors associated with a poorer prognosis include Male sex, old age, severe symptoms of Heart Failure, Hypotension, Acute coronary syndrome, elevated plasma BNP concentration, hyponatremia, and renal function impairment. [23][24][25][26]

3.6.2 CONCLUSION

The number of people living with Heart Failure syndrome is set to increase steeply in the next decades because of an improving prognosis and a rapidly aging population. The typical patient is elderly, as likely to be female as male, and will be suffering from considerable comorbidities. The direct cost to the health care system is substantial and is largely driven by hospitalization costs. The epidemiology suggests that efforts to prevent and treat hypertension

and the other risk factors for coronary artery disease (including obesity, diabetes, hyperlipidemia, and cigarette smoking) will at least delay, if not prevent, the development of heart failure. Once the syndrome develops, the prognosis is poor, particularly in the first months after diagnosis, but has improved substantially in the past decades, presumably as a result of better diagnosis and therapy with angiotensin-converting enzyme inhibitors and -blockers. The epidemic of heart failure is already on us and will be a challenge for all health care settings for the foreseeable future. Management of Diastolic Heart Failure remains empirical and should be oriented towards reduction filling pressures of ventricles, at the same time not reducing the cardiac output and achieving control of symptoms

Abnormalities of diastolic function that range from asymptomatic heart disease to overt heart failure are common in hypertensive patients. An early consequence in systemic hypertension is LV Diastolic dysfunction which is exacerbated in ischemia and left ventricular hypertrophy. Diastolic function is most accurately measured by Cardiac catheterization. Alternatively, non-invasive methods like echocardiography and radionuclide angiography are used.

The emergence of this “new” form of HF engendered considerable early skepticism, despite growing epidemiologic evidence of its importance. Controversy about the significance of HFnlEF has largely but not completely abated. Our understanding of this syndrome is incomplete and will continue to evolve as the field advances.

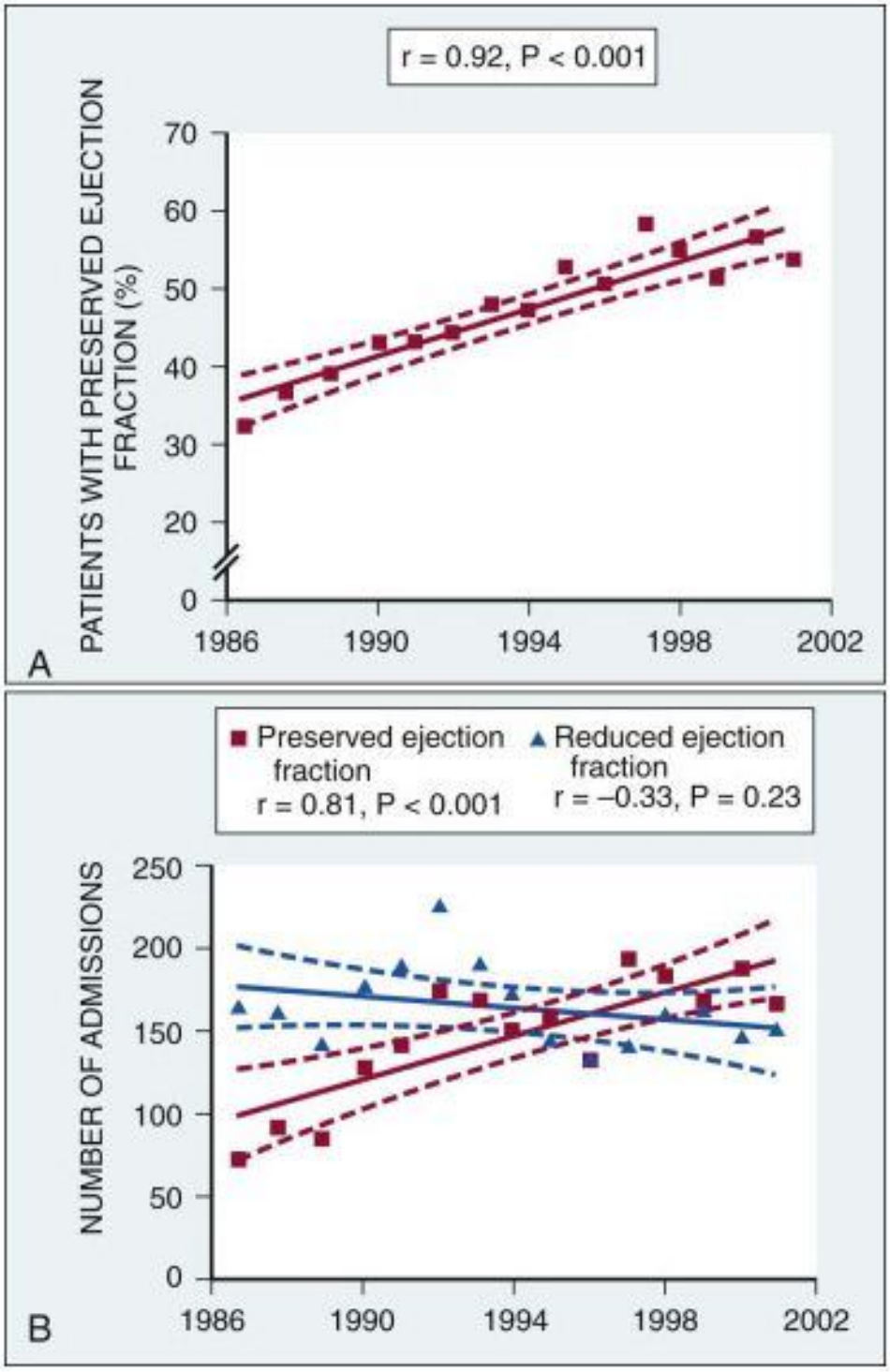


FIG 3.6.1 SHOWING INCREASED PREVALENCE OF HFNLEF[27]

The term HFnlEF has been used in current HF management guidelines.[28,29] Numerous epidemiologic studies and national HF registries have defined the prevalence of HFnlEF in various HF populations and have documented a prevalence of 50% to 55%.[30,31-34] The prevalence of HF increases with age and similar in men and women (Fig 3.6.2). The prevalence of HF with a depressed EF increases with age but is more common in men than in women at any age (Fig 3.6.2), whereas the prevalence of HFnlEF increases even more dramatically with age (more than HF with a reduced EF) and is much more common in women than in men at any age (Fig 3.6.2)[35]

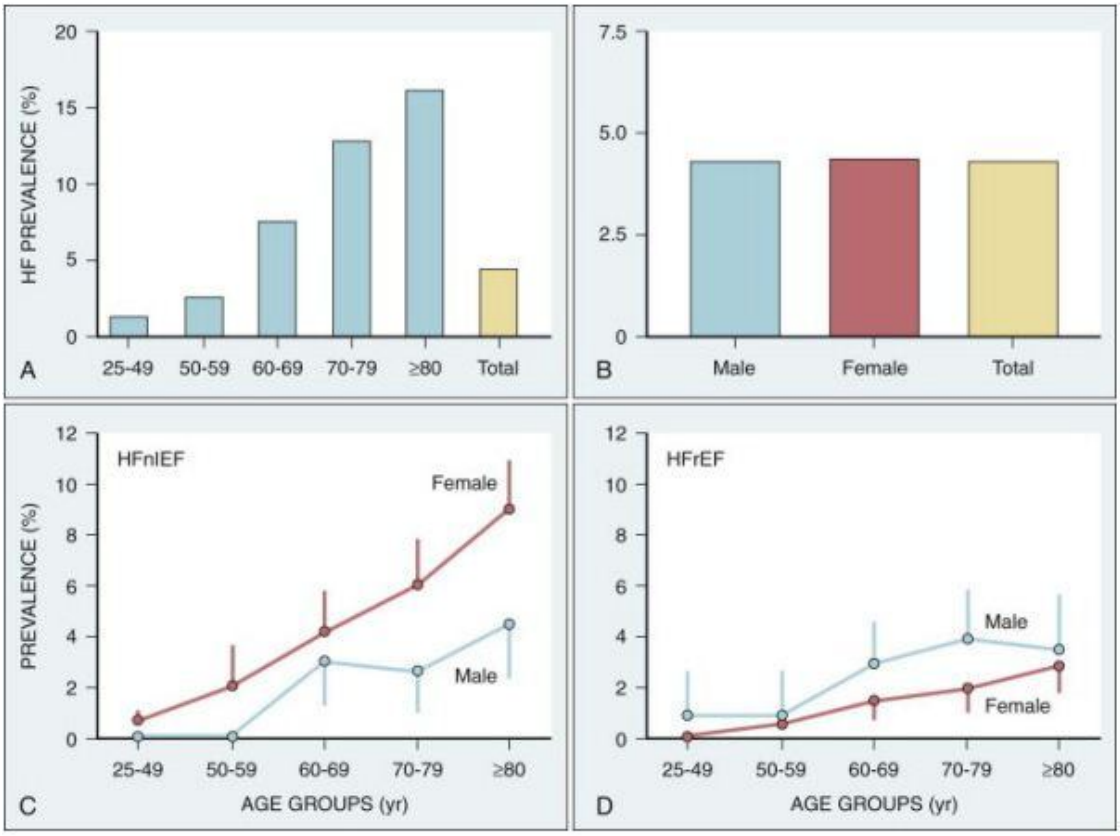


FIG 3.6.2 PREVALENCE CHARACTERISTICS OF HFNIEF

3.6.3 Aging

Although cardiovascular disease may contribute to diastolic dysfunction in older people, studies have also suggested that diastolic function deteriorates with normal aging.[36] Structural cardiac changes with aging (e.g., increased cardiomyocyte size, increased apoptosis with decreased myocyte number, altered growth factor regulation, focal collagen deposition) and functional changes at the cellular level involving blunted beta-adrenergic responsiveness, excitation-contraction coupling, and altered calcium-handling proteins may contribute to diastolic dysfunction with normal aging. Some studies have suggested that prolonged, sustained endurance training may preserve LV compliance with aging and help prevent HF in the elderly.[37]

3.6.5 Gender

Along with age, female gender is a potent risk factor for HFnlEF .Indeed, there appear to be important age-gender interactions, such that the HFnlEF prevalence increases sharply with age in women than prevalence of Heart Failure with a reduced EF.[38] The reasons for the female prominence in HFnlEF are not entirely clear, but women have higher vascular and LV systolic and diastolic stiffness than men do, and vascular and ventricular stiffness increases more dramatically with age in women.[39] Unique coronary vascular

functional changes in women may also play a role in the pathophysiologic process of HFnlEF.

3.6.6 Hypertension

Hypertension is the most commonly associated cardiac condition in patients with HFnlEF. Chronically increased blood pressure is an important stimulus for cardiac structural remodeling and functional changes. The resultant hypertensive heart disease is characterized by LV hypertrophy (LVH), increasing vascular and ventricular systolic stiffness, impaired relaxation, and increased diastolic stiffness, all factors linked to the pathogenesis of HFnlEF.[40] In the presence of hypertensive heart disease, ischemia produces exaggerated increases in filling pressures, and hypertensive heart disease and ischemic heart disease are often present in combination in patients with HFnlEF. Elucidating which factors mediate transition to HFnlEF in systemic hypertension needs further active investigation.

3.6.7 Coronary Artery Disease

The reported prevalence of coronary artery disease or myocardial ischemia in patients with HFnlEF varies widely

3.6.8 Atrial Fibrillation and Other Rhythm Disturbances

Whereas atrial fibrillation may cause decompensated HF in patients with diastolic dysfunction, diastolic dysfunction (in the absence of HF) is also a risk factor for atrial fibrillation.[41] Thus, diastolic dysfunction, atrial fibrillation, and HFnlEF are common and related conditions that probably share common pathogenic mechanisms in the elderly.

3.6.9 Obesity

The chance of developing Heart Failure is increased in obesity In general, patients with HFnlEF are more often obese than. Obese individuals have increased prevalence of LV diastolic dysfunction is increased in obese persons. Increased adiposity not only imposes an adverse hemodynamic load on the heart but also is a source of a large number of biologically active peptide and nonpeptide mediators, many linked to chronic inflammation. Studies using tissue Doppler imaging or invasive LV pressure measurement have reported an association between diastolic dysfunction, elevated filling pressures, and obesity, even in the absence of a diagnosis of HF.[42]

3.6.10 Sleep Apnea

Obstructive sleep apnea is common in patients with HFnlEF risk factors (obesity, atrial fibrillation, and hypertension) and in HFnlEF and can contribute to symptom severity and probably promote progression of HF. Central sleep apnea can occur in association with severe HFnlEF.

3.6.11 Rarer Causes of Heart Failure with a Normal Ejection Fraction

Hypertrophic cardiomyopathy, infiltrative cardiomyopathy such as amyloidosis, valvular disease, and constrictive Pericarditis should always be considered in young patients with HFnlEF or patients with other suggestive features.

3.7 LEFT VENTRICULAR HYPERTROPHY AND HYPERTENSION

Left Ventricular Hypertrophy in systemic hypertension is usually “concentric” hypertrophy with myofibrils showing circumferential hypertrophy, with contractility either normal or increased, wall thickness relatively increased, and end-diastolic volumes either normal or low, relaxation being impaired (“diastolic dysfunction”).

Cardiomyocyte hypertrophy and cardiac fibrosis are the hallmarks of pathologic LVH due to pressure overload. LVH in hypertensive patients is

independent and powerful predictor of morbidity and mortality. The structural abnormalities in the hypertensive heart extend beyond myocyte hypertrophy and include medial hypertrophy of the intramyocardial coronary arteries and collagen deposition leading to cardiac fibrosis.[43] These changes result from pressure overload and the neurohormonal activation that contributes to hypertension. LVH is also contributed by both hemodynamic and genetic factors

On the electrocardiogram, LVH with strain is a serious harbinger of new-onset heart failure and heart failure death.[44] Echocardiography detects LVH more sensitively than electrocardiography does. Whereas electrocardiographic LVH is present in 5% to 10% of hypertensives, echocardiographic LVH is present in nearly 30% of unselected hypertensive adults and in up to 90% of patients with severe uncontrolled hypertension. Cardiac magnetic resonance imaging (MRI) is even more sensitive. [45]

3.7.1 IMPAIRED CORONARY VASODILATOR RESERVE

Even in the absence of atherosclerosis, the hypertensive heart has blunted or absent coronary vasodilator reserve, leading to subendocardial ischemia under conditions of increased myocardial oxygen demand. The combination of subendocardial ischemia and cardiac fibrosis impairs diastolic relaxation, leading to exertional dyspnea and diastolic heart failure. About 30- 50% of

persons with stage 1 and 2 systemic hypertension have impaired LV relaxation ans more than two-thirds of persons with severe hypertension have abnormal LV relaxation.[46]

Echocardiography is a more sensitive technique than electrocardiography for LVH detection. Persons with LVH are twice more likely to suffer from premature cardiovascular events or death. Systemic Hypertension if aggressively controlled can reverse or regress LVH and thereby reduce cardiovascular disease risk. ACE inhibitors have shown to achieve consistent reduction in left ventricular mass. In LIFE study, LVH was found to be significantly reduced by by a losartan-based based regimen.[47]

3.8 LARGE-VESSEL DISEASE & HYPERTENSION

Hypertension constitutes a major risk factor for aortic dissection. Systemic Hypertension is present in the overwhelming majority of patients with aortic dissection (distal more than proximal dissection), abdominal aortic aneurysm, and peripheral arterial disease. One-time abdominal ultrasound screening for abdominal aortic aneurysm is recommended after the age of 65 years in smokers and in those with severe systolic hypertension, and it should be performed if aortic pulsations are detected below the umbilicus because most abdominal aortic aneurysms occur below the origin of the renal arteries.

In adults, a diameter of 2.1 cm/m^2 has been considered the upper normal range in ascending aorta and above this value, aortic root dilatation is said to be present. Aortic root dilatation occurs at the sinuses of Valsalva.[48]. Trans-oesophageal echocardiography is technique of choice for measurement of arch of aortic and descending aorta due to better visualization

3.9 HYPERTENSION AND AGE:

Systemic Hypertension increases in prevalence along with advancing age. More than 50% of persons in 60–69 years of age and three-fourths of those in 70 years of age and older are affected.[49] Lifetime risk of systemic hypertension was found to be 90 % for non-hypertensiv men and women at 55 or 65 years who survived to 80–85years of age. [50] Pulse pressure and SBP are powerful predictors of cardiovascular disease than DBP in older individuals

3.10 HYPERTENSION IN WOMEN

Females exhibit sexual dimorphism in Blood pressure. SBP levels were lower in early adulthood Women, while DBP levels were lower in sixth decade. DBP is marginally lower in women regardless of age.[51] Systemic hypertension is also less prevalent in women than men. But, hypertension incidence occurs more rapidly in women after the fifth decade and is either equal or exceeds men in the sixth decade of life.

3.11 HYPERTENSION AND OBESITY

Across various populations, hypertension prevalence increases linearly with average body mass index. More than half of the cases of hypertension may be due to obesity

Indians have unique features with regard to obesity like abdominal adiposity, increase in intra- abdominal and subcutaneous fat, and fat deposition in ectopic sites like liver, muscle, etc. Obesity is associated with widely prevalent type 2 diabetes mellitus and metabolic syndrome in Asian Indians. Based on percentage body fat and morbidity data, The limits of normal BMI were narrower and lower in Asian Indians than white Caucasians on the basis of percentage of body fat and morbidity data. Consensus Statement for Diagnosis of Obesity in asian Indians states Normal BMI: 18.0-22.9 kg/m2, Overweight: 23.0-24.9 kg/ m2, Obesity: >25 kg/m2.[52]

Reduction of age related rate of weight gain is the major goal of management of both the metabolic syndrome and overweight and obesity.

3.12 TOBACCO AND HYPERTENSION

Cigarette smokers have a higher chance of developing large vessel as well as small vessel disease. Nearly 90% of peripheral vascular disease and 50% of aortic aneurysms are attributed to smoking. Also 20–30% of CAD and 10% of stoke are due to cigarette smoking. Cigarette smoking also interacts with other cardiac risk factors and leads too increment in risk of CVD

Risk of second coronary event is reduced within 6-12 months of cessation of cigarette smoking. Within first few years of cessation of smoking , risk of first MI and death due to CAD also decline After 15 years of abstinence, Risk of a new myocardial infarction or death from coronary heart disease in former smokers is similar to non- smokers after 15 years of cessation.

3.13 IMPROVING HYPERTENSION CONTROL

Systemic hypertension remains an inadequately treated condition, even though it is the most common indication for adults to visit a physician,.[53] hypertension therapy will be most effective only if the patient is well motivated to take anti hypertensive medications and maintain health-promoting lifestyle.[54]

Clinical inertia pn the part of the physician must be overcome. [55]

Physicians must periodically review their patient files and assess degree of compliance and achievement of established goals and treatment. Patients needed to informed about their BP during each visit and a written record has to be given to them.Physicians should work with other health care professionals in order to influence improvement in patient lifestyles and BP control.

Patient attitudes are greatly influenced by cultural differences, beliefs, and previous experiences with the health system.[56] Attitudes of hypertensive patients should be understood so as to build trust and communication with patients and their families, as cultural beliefs, previous hospital experience greatly influence patient attitudes. Furthermore, behind the inherent nature of the disease lurks another disquieting feature of the therapy of most hypertension; it may not benefit the majority of patients who adhere faithfully to their treatment.[57]

The situation may change for at least three reasons. First, significant increases in cardiovascular morbidity and mortality have now been amply documented, even in those with levels of blood pressure below 140/90 mm Hg.[58] Second, blood pressure levels above the assumed normal (i.e., 120/80 mm Hg) interact with other cardiovascular risk factors, calling for the need to address not only blood pressure but the patient's multiple risk factors.[59] Third, and perhaps most importantly, treatment with drugs that have almost no side effects can delay, if not prevent, the progression of prehypertension into overt hypertension.[60] Beyond these reasons, the potential for an inexpensive polypill with antilipemic, antihypertensive, and antithrombotic components may soon be realized.[61] All these reasons are amplified by the realization that the prevalence of hypertension continues to increase as the population grows fatter and older.

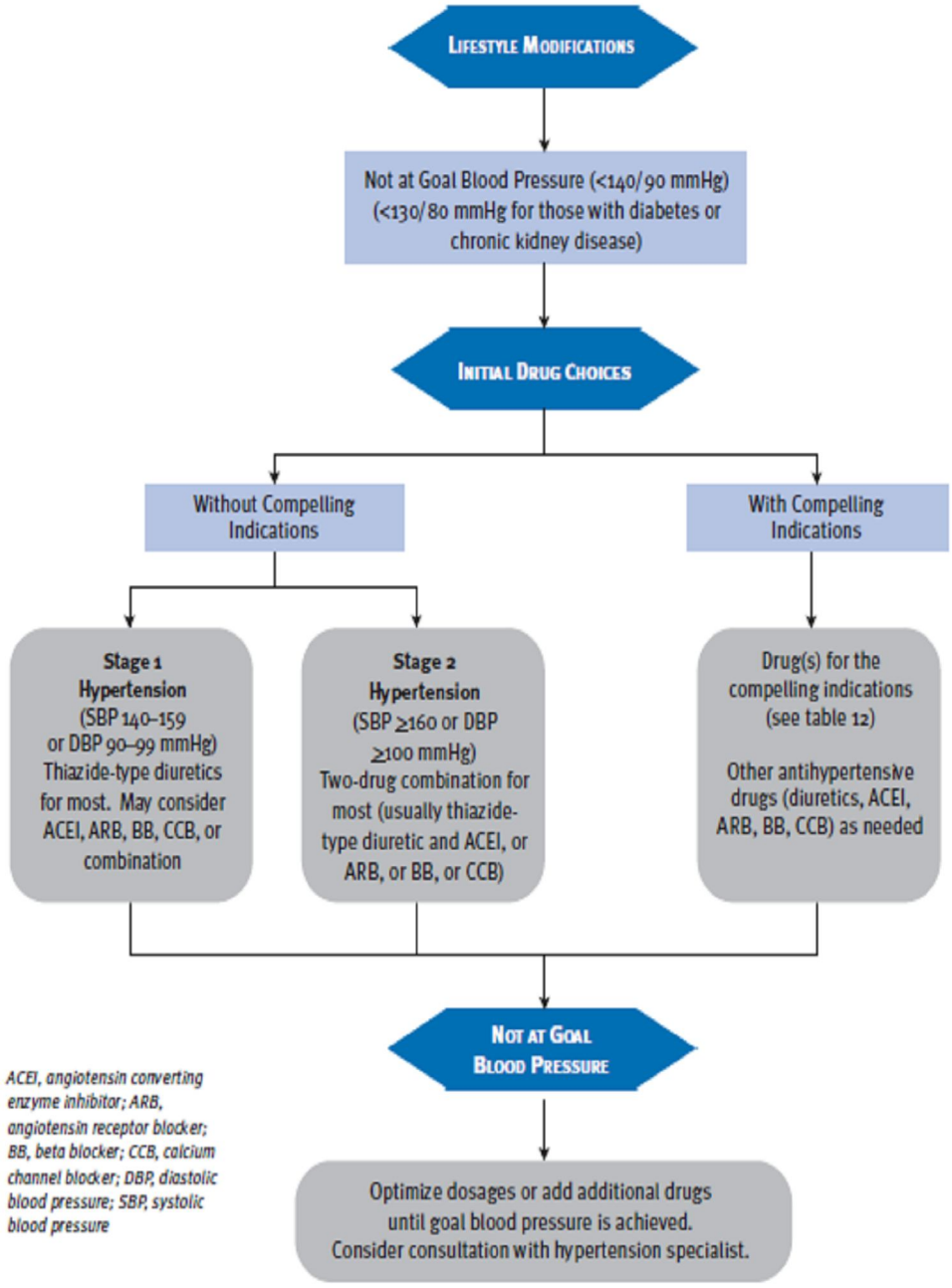


FIG 3.13.1[4] ALGORITHM FOR TREATMENT OF HYPERTENSION

3.14 FOLLOWUP AND MONITORING

Once antihypertensive drug therapy is initiated, most patients should return for follow-up and adjustment of medications at monthly intervals or until the BP goal is reached. More frequent visits will be necessary for patients with stage 2 hypertension or with complicating comorbid conditions. Serum potassium and creatinine should be monitored at least one to two times per year. After BP is at goal and stable, follow-up visits can usually be at 3- to 6-month intervals.

Comorbidities such as HF, associated diseases such as diabetes, and the need for laboratory tests influence the frequency of visits. Other cardiovascular risk factors should be monitored and treated to their respective goals, and tobacco avoidance must be promoted vigorously. Low-dose aspirin therapy should be considered only when BP is controlled because of the increased risk of hemorrhagic stroke when the hypertension is not controlled.[62]

3.15 ECHOCADIOGRAPHY IN HYPERTENSION

Echocardiography remains the most frequently used and usually the initial imaging test to evaluate all cardiovascular diseases related to a structural, functional, or hemodynamic abnormality of the heart or great vessels. Even with advances in other cardiovascular imaging modalities, such as cardiac magnetic resonance imaging and computed tomography. Whereas electrocardiographic

LVH is present in 5% to 10% of hypertensives, Echocardiographic LVH is present in nearly 30% of unselected hypertensive adults and in up to 90% of patients with severe uncontrolled hypertension.

3.15.1 PRINCIPLE

Echocardiography uses ultrasound beams reflected by cardiovascular structures to produce characteristic lines or shapes caused by normal or altered cardiac anatomy in one, two, or three dimensions by M (motion)–mode, two-dimensional, or three-dimensional echocardiography, respectively. Doppler examination and color flow imaging provide reliable assessment of cardiac hemodynamics and blood flow.[65]

Transesophageal echocardiography (TEE) has markedly improved resolution of echocardiographic images, including tissue Doppler imaging, three dimensional imaging and strain imaging and has enhanced the ability to assess systolic and diastolic function.[66]

Reliable noninvasive hemodynamic evaluation and confident delineation of cardiovascular structures by echocardiography have dramatically reduced the clinical necessity for hemodynamic cardiac catheterization. Increasingly, patients undergo valvular or congenital heart surgery on the basis of an echocardiographic diagnosis. Echocardiographic units are also being miniaturized to become an extension of a clinician's physical examination.[67] In

our opinion, the appreciation of cardiac anatomy and hemodynamics by bedside echocardiography makes a physician's clinical evaluation, including physical examination, more relevant to the care of patients. For all physicians who care for patients with a cardiovascular problem, it is essential to know how echocardiographic images are obtained, what type of information echocardiography can provide, and how it should be used for management.

Real-time two-dimensional echocardiography produces high-resolution images of cardiac structures and their movements. The images are obtained from four standard transducer locations by manual rotation and angulation of the transducer and quantitative and qualitative measurements regarding cardiac dimensions are obtained. [68] 2D echocardiography also helps in providing framework for Doppler examination and color flow imaging. With advances and clinical experience in three-dimensional and multidimensional echocardiographic imaging, visualization and quantitation of cardiovascular structure, function, and hemodynamics will improve. It may be feasible in the near future for echocardiogram to begin with three-dimensional images.

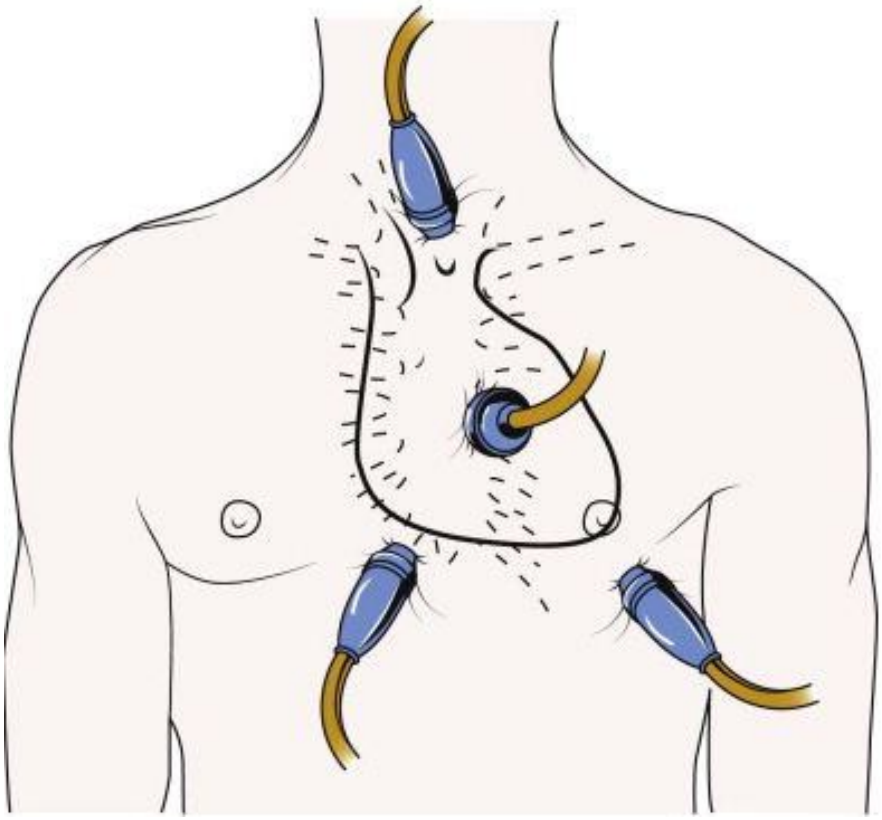


FIG 3.15.1- FOUR STANDARD TRANSDUCER POSITIONS—PARASTERNAL, APICAL, SUBCOSTAL, AND SUPRASTERNAL—USED TO VISUALIZE THE HEART AND GREAT VESSELS.

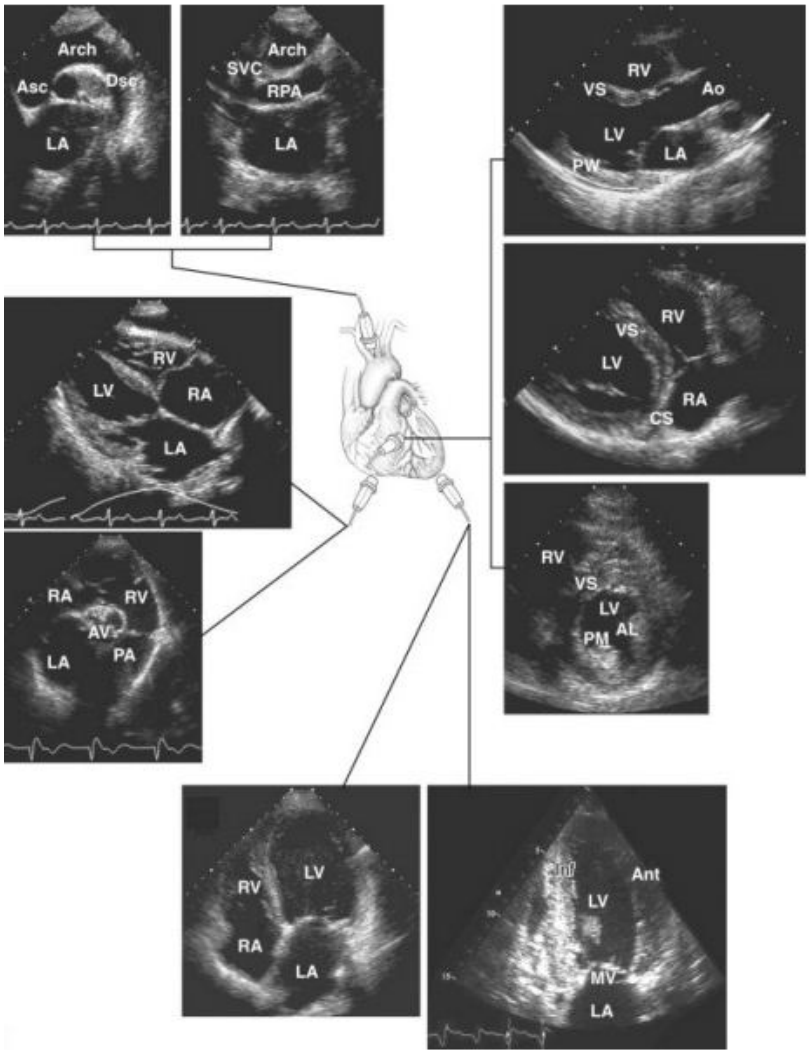


FIG 3.15.2 – VARIOUS TOMOGRAPHIC ECHOCARDIOGRAPHIC IMAGES [69]

3.15.2 M-MODE ECHOCARDIOGRAPHY

An M-mode recording is used to record the motion of cardiac structures. Primary use of M-mode is display of subtle abnormalities of cardiac motion, timing of cardiac event and measuring chamber size. Thus, it is important to recognize the characteristic M-mode features to understand the pathophysiologic mechanisms of cardiovascular disorders.

3.15.3 DOPPLER ECHOCARDIOGRAPHY

Doppler echocardiography measures blood flow velocities on the basis of the Doppler Effect. Frequency of reflected ultrasound waves (*f*r) increases when the red blood cells are moving toward the source of ultrasound and decreases when the red blood cells are moving away. The difference in frequency between transmitted sound and reflected sound is the frequency shift or Doppler shift: ($\Delta f = f\text{r} - f\text{o}$). Once Δf is calculated, the velocity of red blood cells IS calculated as $v = \Delta f \times c/2 f\text{o}$ (c - speed of sound in blood (1540 m/sec). Blood flow velocities measured with Doppler echocardiography are used to derive various hemodynamic data.

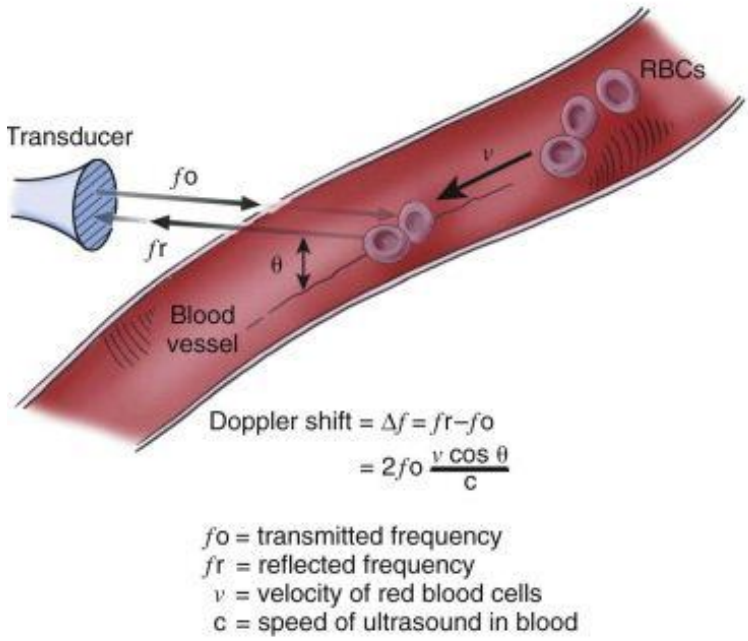


FIG 3.14.3 DIAGRAM OF THE DOPPLER EFFECT [70]

3.15.4 COLOR FLOW IMAGING

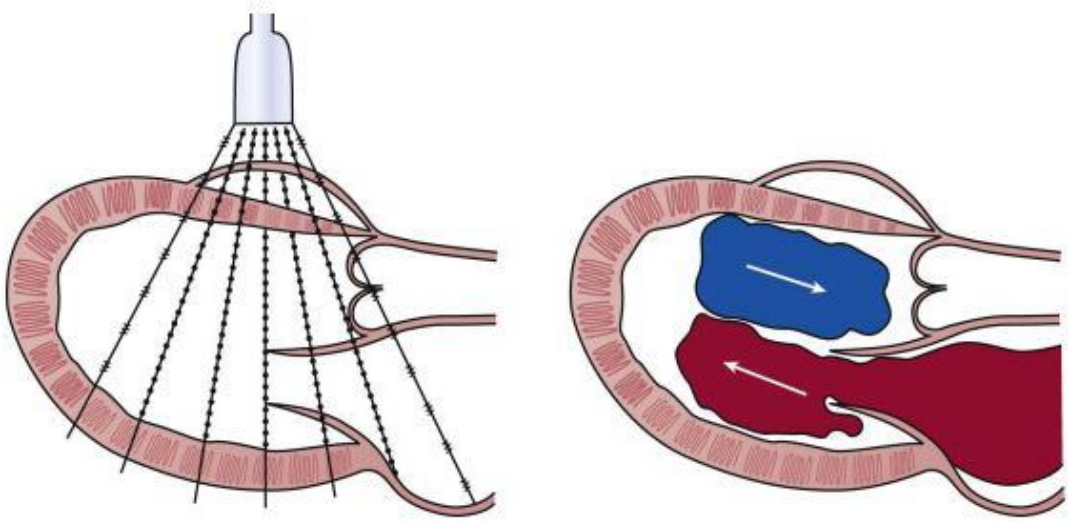


FIG 3.14.4 DRAWING ILLUSTRATING HOW COLOR FLOW IMAGING IS PERFORMED AND DISPLAYED[71]

Color Doppler is a form of pulsed-wave Doppler displaying blood flow or myocardial velocities in various colors (usually red, blue, and green) or their combinations, depending on turbulence, velocity, direction. During intracavitary flow, blood flow is directed toward the transducer and has positive frequency shift and color is coded in red shades, whereas blood flow directed away from transducer has negative frequency shift and color is coded in blue shades. Abnormal blood flow is easily recognized by combinations of multiple colors according to the directions, velocities, and degrees of turbulence. The width and size of abnormal intracavitary blood flows are used to evaluate the degree of

valvular regurgitation or cardiac shunt. Color Doppler is used also for determination of diastolic function and timing of intracardiac events.

3.15.5 TISSUE DOPPLER AND STRAIN IMAGING

Tissue Doppler imaging (TDI) records the motion of tissue or other structures with a velocity or frequency shift much lower than that of blood flow. Doppler echocardiography for blood flow measures the velocities of red blood cells (velocity usually higher than 20 cm/sec and up to 800 cm/sec in the case of valvular disease). However, the velocities of myocardial tissue are much lower (<30 cm/sec) but with larger amplitudes than those produced by blood. Therefore, pulsed-wave Doppler was modified to record the low velocities of myocardial tissue and to reject the high velocities generated by blood flow. TDI can also be displayed in color mode. A major limitation of tissue velocity recording is that tissue velocities measured by TDI may overestimate or underestimate the active component or function of the tissue because of translational motion or tethering, respectively. Strain (ϵ) and strain rate imaging can overcome this limitation by measuring the actual extent of stretching or contraction

Strain imaging also allows left ventricular (LV) rotational motion, often referred to as torsion or twist, to be assessed. The spiral shape of LV myocardial fibers results in a complex three-dimensional torsion mechanism for systolic

contraction and untwisting for diastolic relaxation.[72] degree of torsion or twisting appears to be related to aging and diastolic function as well as systolic contraction.

The clinical applications of TDI and strain imaging are increasing and provide incremental diagnostic and prognostic value over standard two-dimensional and blood pool Doppler echocardiography. They have been used successfully in assessing regional and global systolic and diastolic function. Myocardial strain becomes abnormal during the early stage of myocardial ischemia as well as in myopathies and appears to be more sensitive for identification of ischemic segments during stress echocardiography.[73-78] TDI is nowadays most commonly used to evaluate diastolic function and diastolic filling pressures.[79,80] Both strain imaging and TDI allow reliable determination of cardiac timing intervals, which is useful in assessing cardiac function[81] and LV intraventricular mechanical dyssynchrony.[82-85]

3.15.6 CHAMBER QUANTITATION

The American Society of Echocardiography (ASE) has published recommendations for chamber quantification with M-mode and two-dimensional echocardiography. It is recommended that the same normal values for chamber dimensions and volumes should be used in both TEE and TTE. [86]

	Women				***Men***			
	REFERENCE RANGE	**ABNORMAL**			**REFERENCE RANGE**	**ABNORMAL**		
		MILDLY	***MODERATELY***	***SEVERELY***		***MILDLY***	***MODERATELY***	***SEVERELY***
Two-dimensional method								
Ejection fraction, %	**≥55**	**45-54**	**30-44**	**<30**	**≥55**	**45-54**	**30-44**	**<30**

TABLE 3.15.1 REFERENCE VALUES OF LEFT VENTRICULAR FUNCTION[87]

MEASURE	*Women*				*Men*			
	REFERENCE RANGE	ABNORMAL			REFERENCE RANGE	ABNORMAL		
		MILDLY	*MODERATELY*	*SEVERELY*		*MILDLY*	*MODERATELY*	*SEVERELY*
Linear method								
LV mass, g	67-162	163-186	187-210	≥211	88-224	225-258	259-292	≥293

TABLE 3.15.2 - REFERENCE VALUES OF LEFT VENTRICULAR MASS AND GEOMETRY[88]

MEASURE	*Women*				*Men*			
	REFERENCE RANGE	ABNORMAL			REFERENCE RANGE	ABNORMAL		
		MILDLY	*MODERATELY*	*SEVERELY*		*MILDLY*	*MODERATELY*	*SEVERELY*
LA area, cm^2	<20	20-30	30-40	>40	<20	20-30	31-40	>40

TABLE 3.15.3- REFERENCE VALUES LEFT ATRIAL AREAS [89]

3.15.7 LEFT VENTRICULAR DIMENSIONS

Usually, LV dimensions are measured from two-dimensional guided M-mode echocardiograms of the left ventricle at the level of mitral leaflet tips or the papillary muscle by the parasternal view. If no extensive regional wall motion abnormalities are present, the LV dimensions measured at the mid-ventricular level can be used to calculate global LVEF. Left ventricular posterior wall and interventricular septum (from the leading edge to the trailing edge) are measured from same M-mode echocardiogram. These values are used to calculate LV mass.

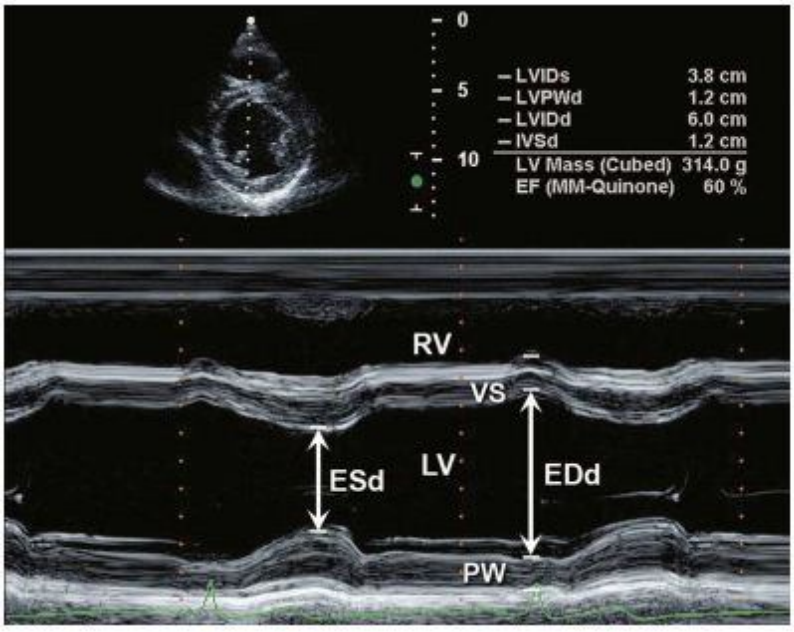


FIG – 3.15.5 TWO-DIMENSIONAL GUIDED M-MODE ECHOCARDIOGRAM OF LEFT VENTRICLE (LV) AT THE PAPILLARY MUSCLE LEVEL.

3.15.8 LEFT ATRIAL SIZE AND VOLUME

Traditionally, LA dimension is determined from parasternal long-axis view during end systole. However, Left atrium size may be underestimated from the parasternal view because there may longitudinal enlargement. Therefore, LA size must be measured from apical views (Tip of mitral valve to posterior wall of LA). However, LA volume is a better measure of LA size and provides better prognostic value. LA size or volume is an important determinant of LA pressure, diastolic function, and prognosis.[90]

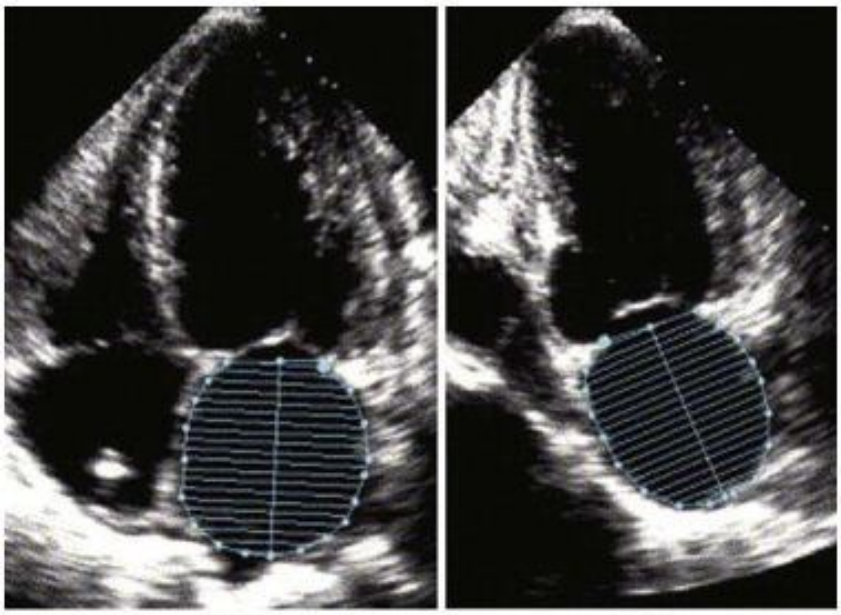


FIG 3.15.6 LEFT ATRIAL VOLUME MEASUREMENT BY BIPLANE AREA-LENGTH METHOD [91]

3.15.9 LEFT VENTRICULAR MASS

Two methods are available for calculation of LV mass from two-dimensional echocardiography: the biplane area-length method and the truncated ellipsoid method. LV mass can also be estimated from measurements of LV dimension and wall thicknesses on two-dimensional or M-mode echocardiograms. Reasonable determination of LV mass in grams is provided by the following formula : $1.04 [(LVID+PWT+IVST)^3 – LVID^3] \times 0.8 + 0.6$ (LVID – LV internal dimension, PWT - posterior wall thickness, IVST - interventricular septal thickness, 1.04 is the specific gravity of the myocardium, and 0.8 being correction factor). . All measurements are made during end diastole.

Three-dimensional imaging has the potential to be the most accurate echocardiographic measure for LV volume and mass. Compared with CMR, Mor-Avi and colleagues [92] showed that interobserver variability was 37% ± 19% of measured LV mass by two-dimensional echocardiography and 7% - 10% by real-time three-dimensional echocardiography.

3.15.10 SYSTOLIC FUNCTIONAL PARAMETERS

Echocardiography can measure several parameters as an expression of systolic function of the heart. These parameters are LVEF, fractional shortening, stroke volume and cardiac index, systolic tissue velocity of the mitral annulus and myocardium, strain, and regional wall motion analysis.

3.15.11 LEFT VENTRICULAR EJECTION FRACTION

The most well accepted expression of global LV function is LVEF. LVEF is a strong predictor of outcome in all major cardiac conditions, although it has many limitations, including load dependency. LVEF is also used to select optimal treatment strategies. In clinical practice, LVEF should be measured more objectively whenever possible, using volumetric measurements as described by the following equation: LVEF = (LVED-LVESV)/LVEDV.

LVEDV and LVESV are LV end-diastolic volume and end-systolic volume, respectively. LVEF can also be calculated from LV dimensions measured with M-mode or two-dimensional echocardiography from the mid-ventricular level using the formula: LVEF = $(LVEDD^2-LVESD^2)/LVEDD^{2.}$ LVEDD and LVESD are end-diastolic dimension and end-systolic dimension, respectively. This equation is actually the percentage change in LV area, or fractional shortening of the LV short axis. Because the apical long axis normally shortens 10% to 15% with systole, an apical correction factor is added on the

basis of the contractility of the apex: 5% to 7% for normal to hyperdynamic apical contraction, 3% for hypokinetic contraction, and 0% for akinetic apex. Because three-dimensional echocardiography can provide LV end-diastolic and end-systolic volumes closer to those measured by CMR, it will become the standard method to calculate LVEF. It can also provide regional LV volume as well as the timing of the smallest volume of each region, which may be helpful in assessing the synchronicity of LV regional contraction.

3.15.12 ASSESSMENT OF DIASTOLIC FUNCTION

Diastolic function assessment must be an integral part in evaluation of cardiac function because about 50% of patients with heart failure have preserved LVEF. Currently, echocardiography is the best noninvasive way to evaluate diastolic function and to estimate filling pressures. M-mode, two-dimensional, and Doppler (blood flow, tissue, and color) echocardiography are all helpful in evaluating diastolic function. Recently, the ASE and European Association of Echocardiography (EAE) released guidelines for assessing diastolic function by echocardiography.[93] The following steps will ensure comprehensive assessment of diastolic function and the identification of heart failure related to diastolic dysfunction:

1. Look for two-dimensional and M-mode echocardiographic evidence suggestive of diastolic dysfunction. Abnormal myocardial relaxation, an

integral part of diastolic dysfunction, decreases the slope (in M-mode) and mitral annulus motion of early diastolic filling and increases LA size. LV wall thicknesses are usually but not necessarily increased

2. Mitral inflow velocities reflect the transmitral pressure gradient, which is usually characteristic of various stages of diastolic dysfunction. Assessment of ventricular compliance is possible from the configuration of mitral inflow velocities. Pulmonary vein flow velocities are also helpful.

3. Myocardial relaxation by TDI can be evaluated. Mitral annulus velocity (e′) during early diastole correlates reasonably well with the status of myocardial relaxation (tau).

4. Mitral inflow velocities (E and A), e′, mitral inflow propagation velocity, and their combination can estimate LV diastolic filling pressure in rest and during exercise.

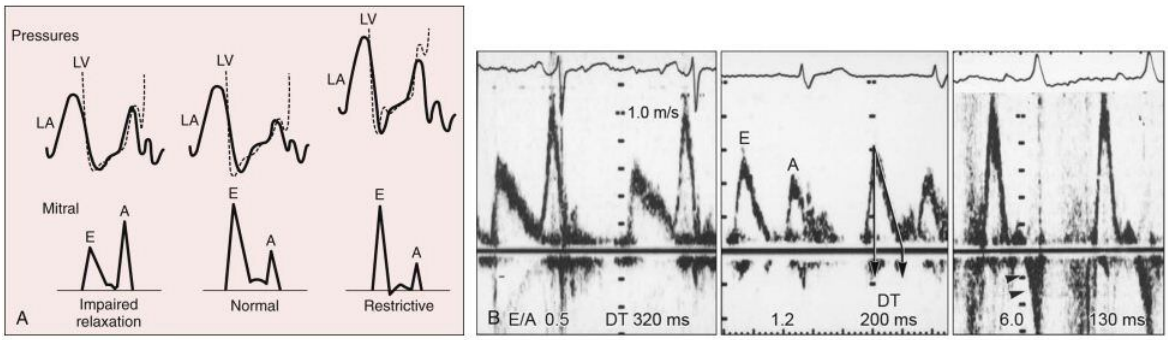


FIG 3.15.7 LV AND LA PRESSURE RELATIONSHIP AND CORRESPONDING MITRAL INFLOW VELOCITIES IN THREE DIFFERENT DIASTOLIC FILLING PATTERNS: IMPAIRED RELAXATION, NORMAL, AND RESTRICTIVE AND ACTUAL DOPPLER RECORDINGS [94]

LV diastolic filling consists of a series of events that are affected by numerous factors, including myocardial relaxation, compliance, cardiac rhythm, and pericardial compliance. In normal diastolic function, adequate filling of ventricles at rest and during exercise without abnormal increase in diastolic pressure or pulmonary venous congestion is ensured. Myocardial relaxation is initial diastolic event.[95]

It is an active energy-dependent process causing LV pressure to decrease rapidly after end of contraction leading to mitral valve opening and rapid early diastolic filling. Elastic recoil due to normal relaxation of LV is a major determinant of early diastolic filling under normal circumstances. Normally, 75% to 80% of LV filling occurs during this phase. During early diastolic

filling, LV pressure continues to decrease until completion of myocardial relaxation (normally about 100 milliseconds) before rising after reaching minimal pressure leading to loss of positive driving force which results in the deceleration of mitral inflow. Later, 20% to 25% of LV filling in normal subjects is by atrial contraction which produces a positive transmitral pressure gradient and inflow.

LV filling during various diastolic phases depends on chamber compliance, elastic recoil, rate of myocardial relaxation, LA pressure, and finally heart rate. The transmitral pressure gradient or the relationship between LA and LV pressures is accurately reflected by mitral inflow Doppler velocities.[96] Diastolic filling is classified initially on the basis of peak mitral flow velocity of early rapid filling wave (E), peak velocity of the late filling wave due to atrial contraction (A), E/A ratio, and MV deceleration time (DT), which denotes time interval for the peak E velocity to reach zero baseline

TDI records the velocity of mitral annulus and has become an essential part of diastolic function evaluation by echocardiography.[97] Radial and circumferential function can also be assessed with speckle tracking strain imaging.[98]

Comprehensive assessment of diastolic filling by echocardiography requires TDI, color M-mode of mitral inflow for propagation velocity, hepatic vein Doppler, pulmonary vein Doppler

3.15.13 GRADING OF DIASTOLIC DYSFUNCTION (OR DIASTOLIC FILLING PATTERN)

The grading of the diastolic filling pattern (or diastolic dysfunction) is based on several parameters. Impaired relaxation is the initial diastolic abnormality. With further progression of disease and a mild to moderate increase in LA pressure, the mitral inflow velocity pattern appears the same as a normal filling pattern (pseudonormalized). With further progression, diastolic filling becomes restrictive. Most patients with restrictive filling are symptomatic and have a poor prognosis unless the restrictive filling can be reversed by treatment. However, restrictive filling may be irreversible and represent the end stage of diastolic heart failure. Therefore, diastolic dysfunction can be graded according to the diastolic filling pattern.[99]

Grade 1 (mild): impaired relaxation with normal filling pressure

Grade 2 (moderate): pseudonormalized mitral inflow

Grade 3 (severe reversible): reversible restrictive (high filling pressure)

Grade 4 (severe irreversible): irreversible restrictive (high filling pressure)

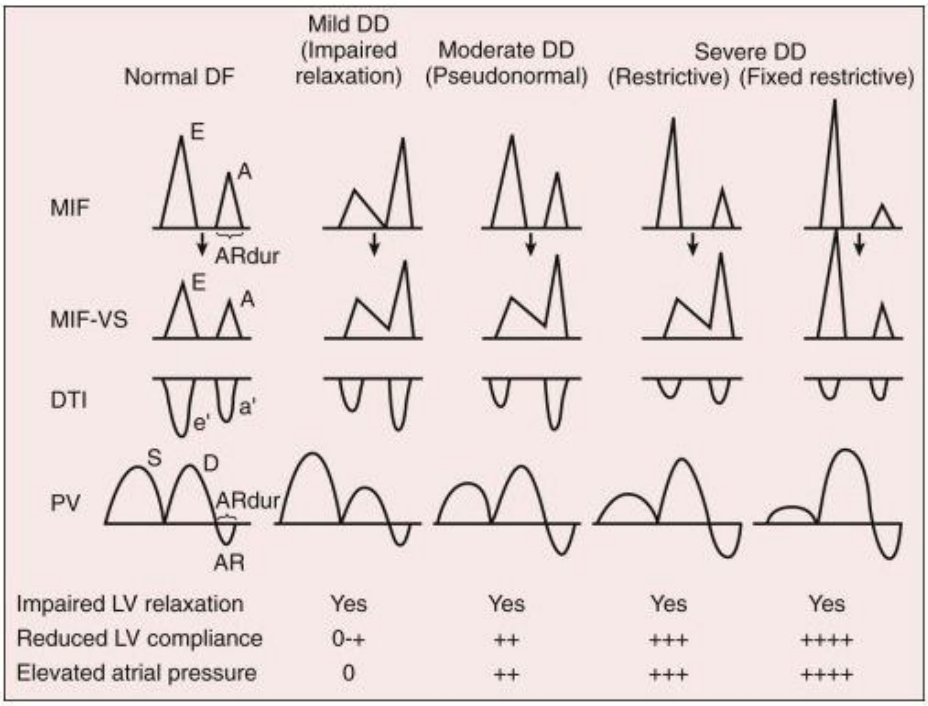


FIGURE 3.15.8 DIAGRAM OF VARIOUS DOPPLER PARAMETERS FOR NORMAL AND ABNORMAL DIASTOLIC FUNCTION (DF) AND (DD)*[100]*

3.15.14 NORMAL DIASTOLIC FILLING PATTERN

In normal young subjects, most filling is completed during early diastole as LV elastic recoil is vigorous. Thus, the E/A ratio is usually 1.5 or higher, DecT is 160 to 240 milliseconds (septal) This vigorous relaxation in normal subjects can also be seen as active motion of the mitral annulus away from the

apex during early diastole in parasternal long-axis and apical four-chamber views.

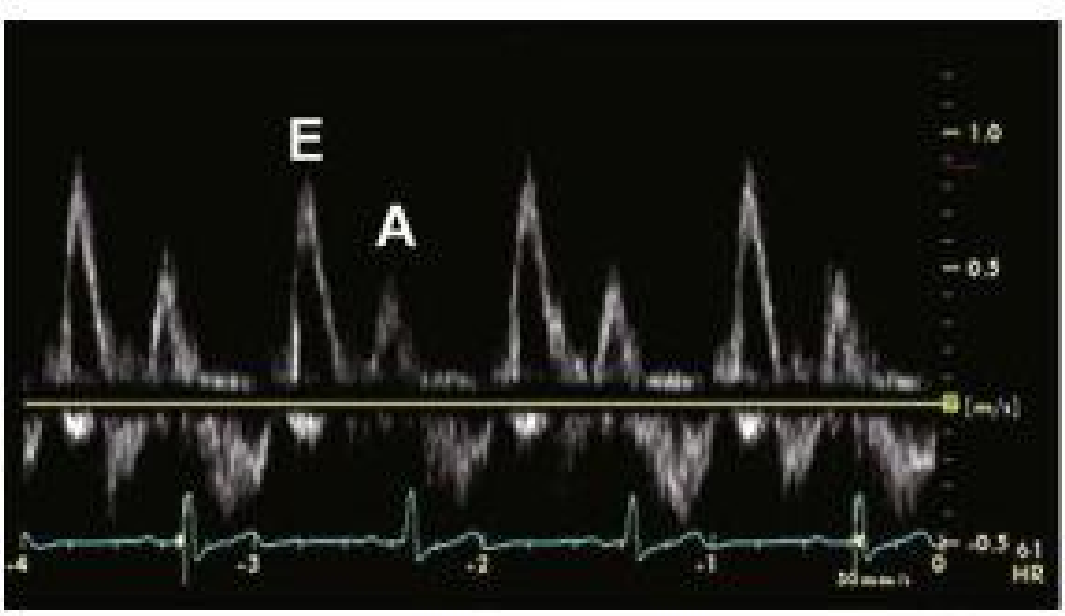


FIGURE 3.15.9 - MITRAL INFLOW FROM A NORMAL SUBJECT

With aging, there is slower decline of LV pressure, and filling becomes slower, producing a diastolic function pattern similar to grade 1 dysfunction. At roughly the age of 65 years, E velocity ia almost equal to A velocity and at more than 70 years of age, E/A ratio is less than 1.0. Similar changes are shown in pulmonary venous flow velocities

In Grade 1 LVDD or mild diastolic dysfunction, there is decrease in Mitral E velocity and increase in A velocity, producing an E/A ratio < 1 and DT is prolonged. Typical cardiac conditions that produce mild diastolic dysfunction are LV hypertrophy, HCM, and myocardial ischemia or infarction as well as aging.

In grade II LVDD or moderate diastolic dysfunction, there is pseudonormalized mitral flow filling pattern and it is better evaluated by TDI.

In Grade 3-4 Diastolic Dysfunction or Severe Diastolic Dysfunction, there is increase in E velocity, decrease in A velocity (but markedly less than E) with an E/A ratio > 2 and DT is shortened (<160 milliseconds)

3.15.15 CLINICAL APPLICATIONS OF DIASTOLIC FUNCTION ASSESSMENT

1. Estimation of filling pressures at rest and with exercise
2. Diagnosis of diastolic heart failure, cardiomyopathy, and constrictive pericarditis.
3. Powerful prognostic indicators for various conditions [101-104]

Therefore, assessment of the diastolic filling pattern allows estimation of LV filling pressures and compliance and relaxation so that optimal treatment strategies can be offered to symptomatic patients with diastolic dysfunction.

MATERIALS AND METHODS

4. MATERIALS AND METHODS

4.1 STUDY DESIGN AND PARTICIPANTS

Patients with systemic hypertension were recruited from Government Royapettah hospital hypertension clinic as outpatients. This study was a single center cross sectional observational study. A total of 100 patients (55 males and 45 females) who met inclusion and exclusion criteria were included in to study after obtaining formal written and oral consent. Patients aged more than 65 years were excluded as age related diastolic dysfunction is more common. Patients aged between 20 and 65 years who had already been diagnosed with systemic hypertension were included. People with history of heart disease and any other comorbid conditions were excluded The ethical committee approval was obtained before starting the study. The results and observations were analyzed statistically. The study was from June 2012 to November 2012

4.2 PROCEDURES AND OUTCOME MEASURES

Blood pressure was measured by the conventional cuff method at the time of echocardiographic examination. Patients with Controlled Blood pressure were defined as those with systolic blood pressure (SBP) < 140 mmHG and diastolic blood pressure (DBP) <90 mmHg . Inadequate BP control was defined as thoses with systolic blood pressure (SBP) ≥ 140 mmHG and/or diastolic

blood pressure (DBP) ≥90 mmHg, as per JNC 7 guidelines. The patient's age, height and weight were also recorded at that time. BMI classification was done according to Consensus Statement for Diagnosis of Obesity, Abdominal Obesity and the Metabolic Syndrome for Asian Indians and Recommendations for Physical Activity, Medical and Surgical Management.[1] As per the consensus Statement , Normal BMI was 18.0-22.9 kg/m2, Overweight: 23.0-24.9 kg/ m2, Obesity: >25 kg/m2 in Asian Indians. Examinations were performed by M-Mode echocardiography and Doppler imaging with the Mind ray® diagnostic ultrasound system to assess cardiac function and structural changes

The following left ventricular structural and aortic parameters were measured by M-mode echocardiography- left ventricular end-diastolic and end-systolic dimension, inter-ventricular septal thickness, posterior wall thickness, at level of chordae tendinae and aortic root diameter was taken at the level of Sinuses of Valsalva. Left ventricular mass using linear method calculated by using the formula : Left ventricular mass = 0.8 (1.04 x $[PW+VS+LVDd]^3$-$LVDd^3$)+ 0.6, where LVDd represents left ventricular end diastolic dimension, VS represents interventricular septal thickness and PW represents posterior wall thickness. LV ejection fraction was determined by using teichholz's method .A threshold of 55% was used to indicate left ventricular systolic dysfunction. Aortic diameter and Left atrial diameter was recorded in apical four chamber view.

The velocity of transmitral blood flow was recorded by conventional Doppler echocardiography for assessing left ventricular (LV) diastolic function. Transmitral blood flow was recorded at the apical transducer position, so that sample volume was situated between the mitral leaflet tips. E/A was determined after recording the peak velocity of early transmitral blood flow (E) and the peak velocity of late transmitral blood flow. Recording of Mitral valve Deceleration time was also recorded. Patients were defined as having aortic root dilatation if the AR(aortic root diameter)/M^2 > 2.1 as per European society of echocardiography guidelines.[2]

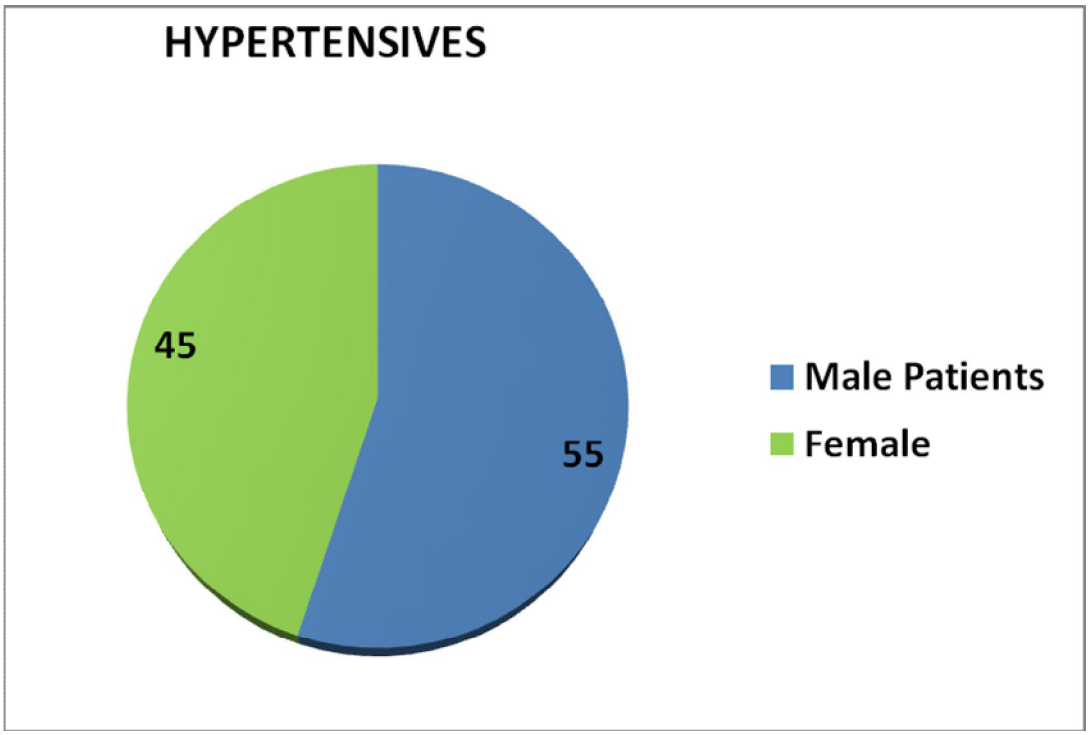
Patients with LV diastolic dysfunction were defined as those with MV (E/A) < 1.0 with prolonged deceleration time (DT) (>240 ms) and those with MV (E/A) > 2.0 with shortened deceleration time (DT) (< 160 ms). [3]

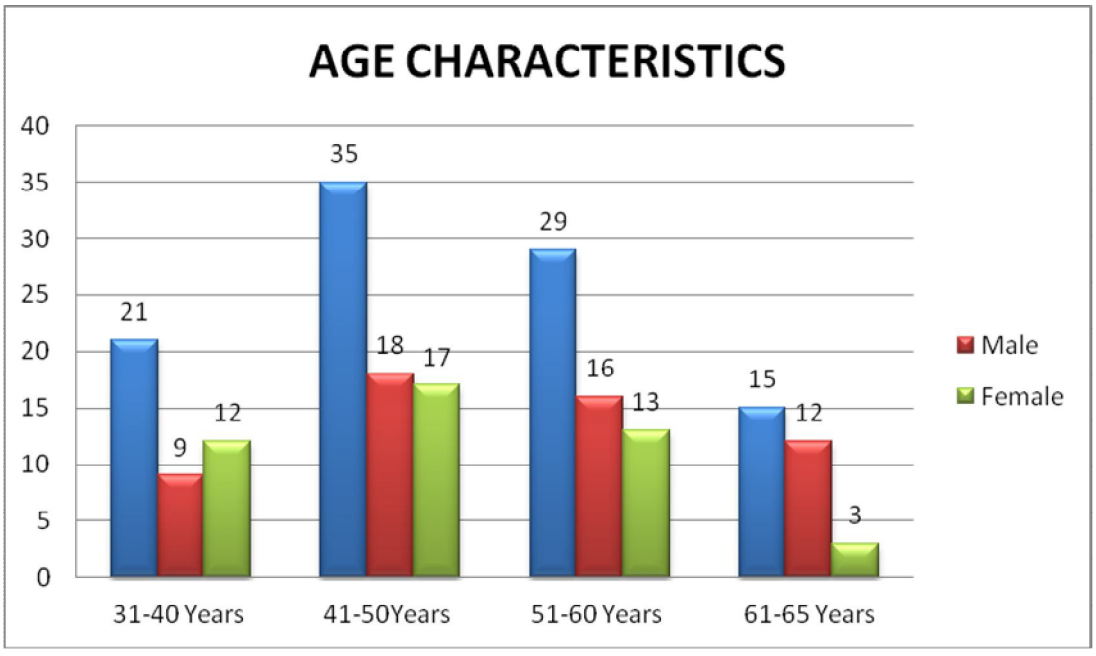
4.3 STATISTICAL ANALYSIS

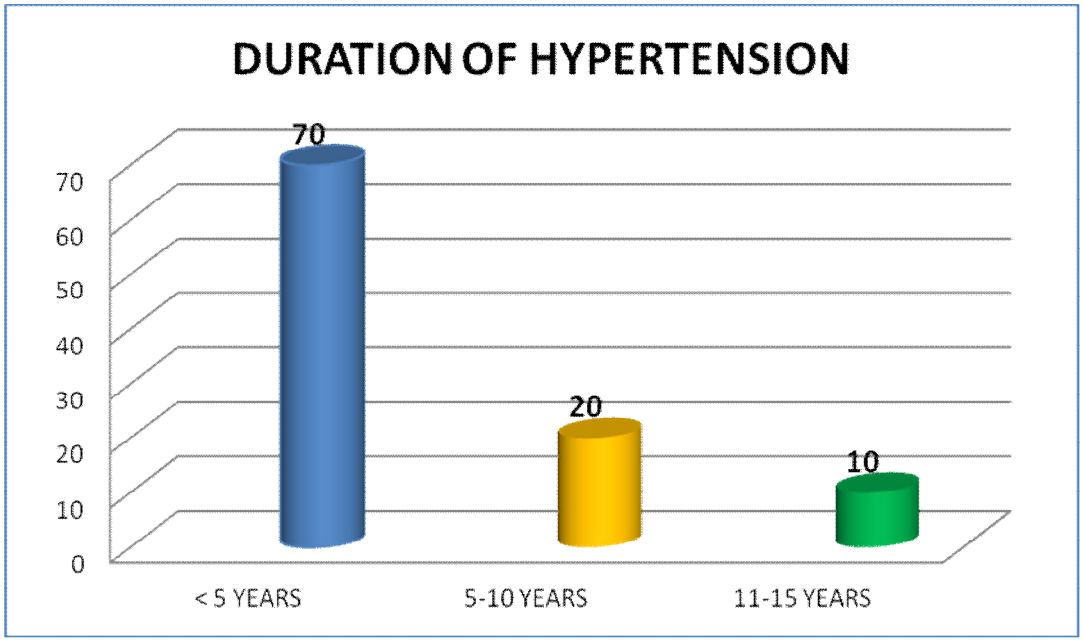
After acquiring the complete set of data and preparing the master chart, statistician working in another facility was approached. The data was analysed with Statistics Products Services Solutions (SPSS)® software for windows with age, sex, duration, control of hypertension, tobacco use, body mass index, left ventricular mass, left ventricular diastolic function, left atrial area and aortic root diameter as variables. A chi squared test was used to analyze the probability of differences between the groups and $p<0.05$ was taken to be statistically significant in all calculations. Graphs were prepared using MS excel software®

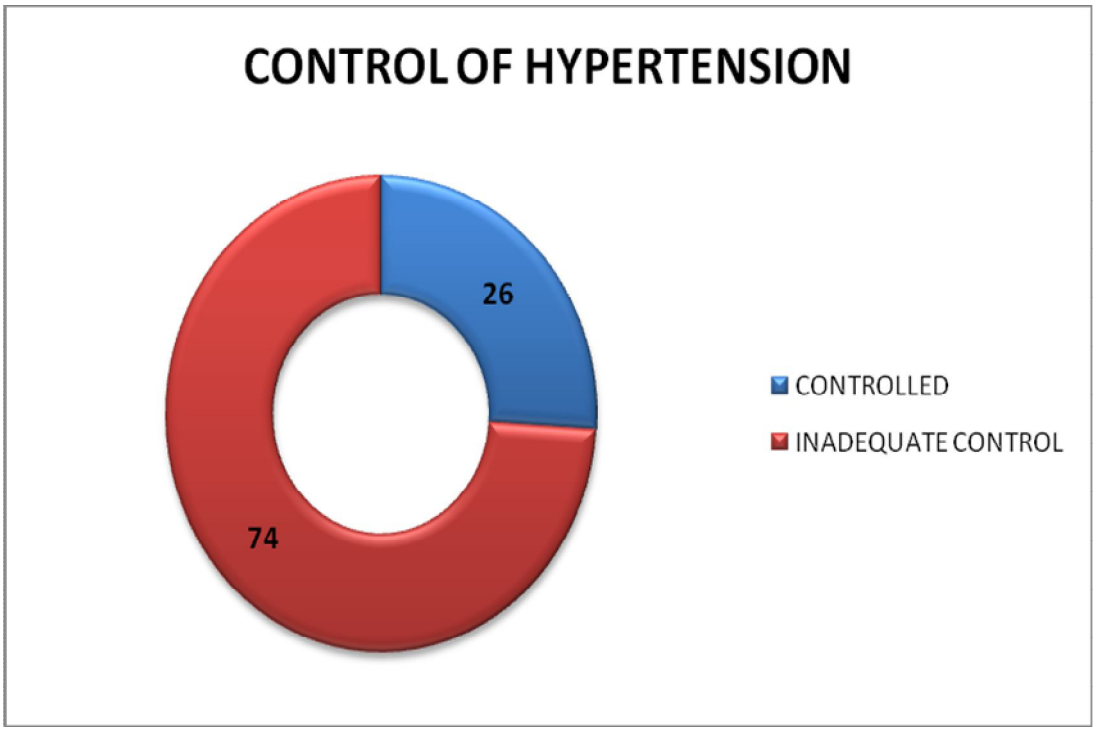
RESULTS

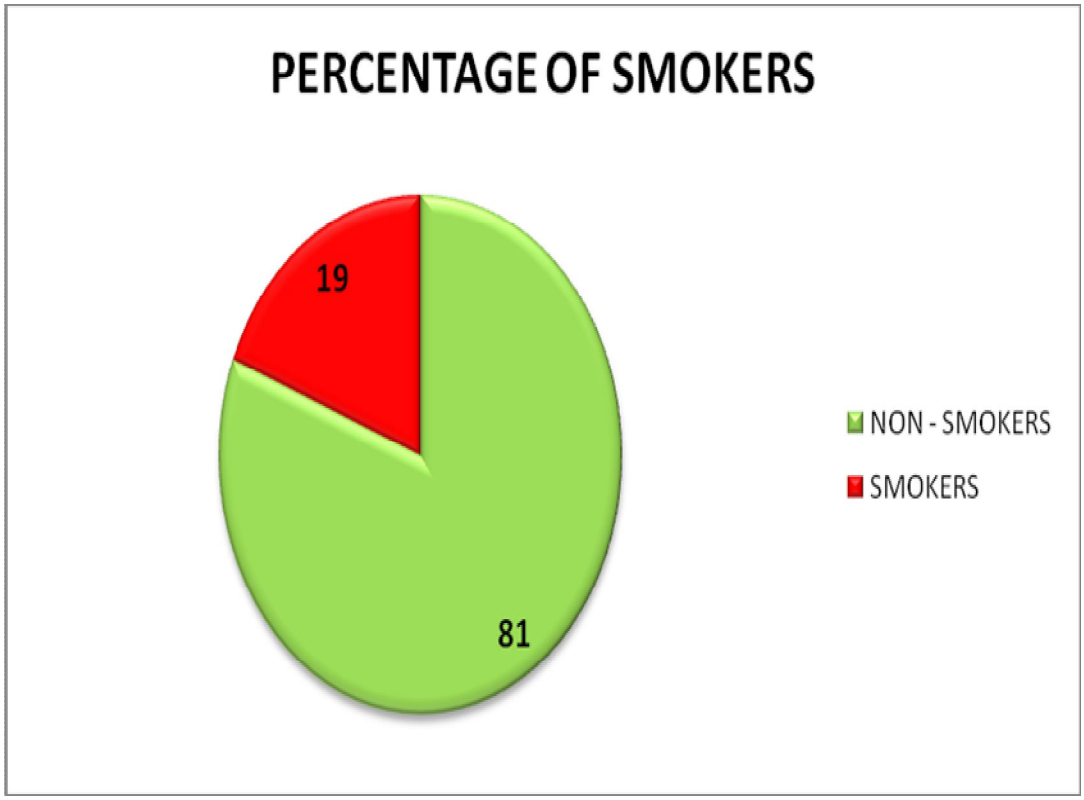
5. RESULTS AND OBSERVATIONS

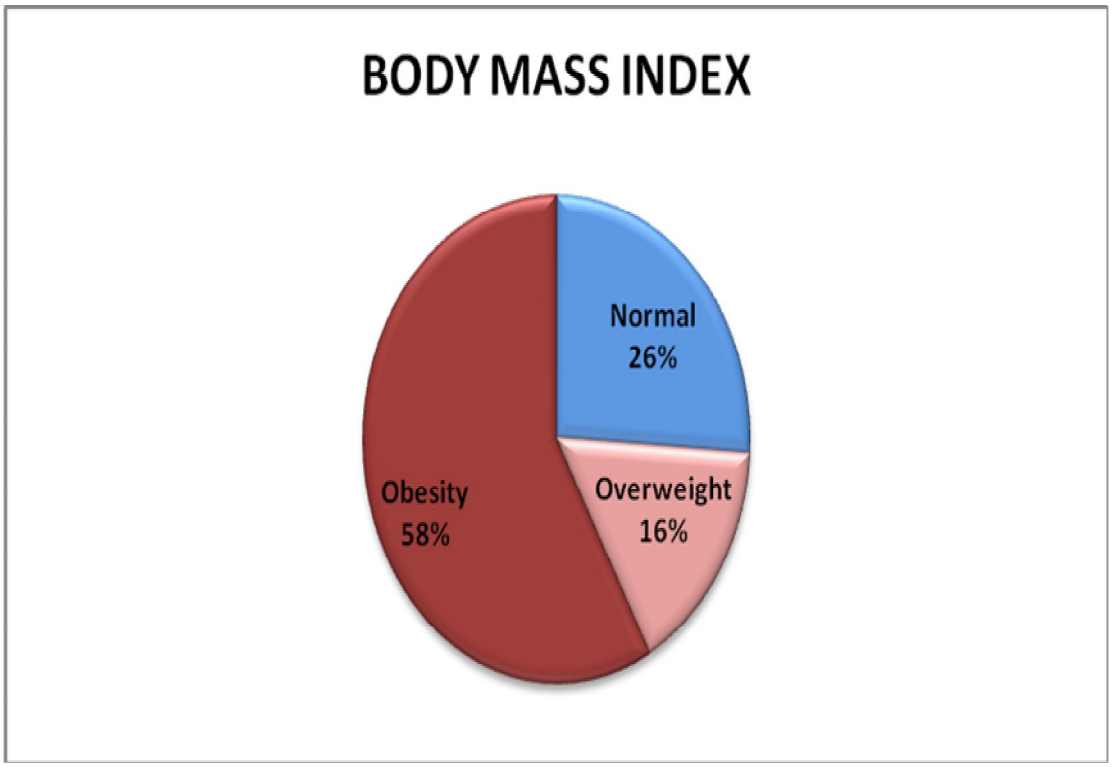




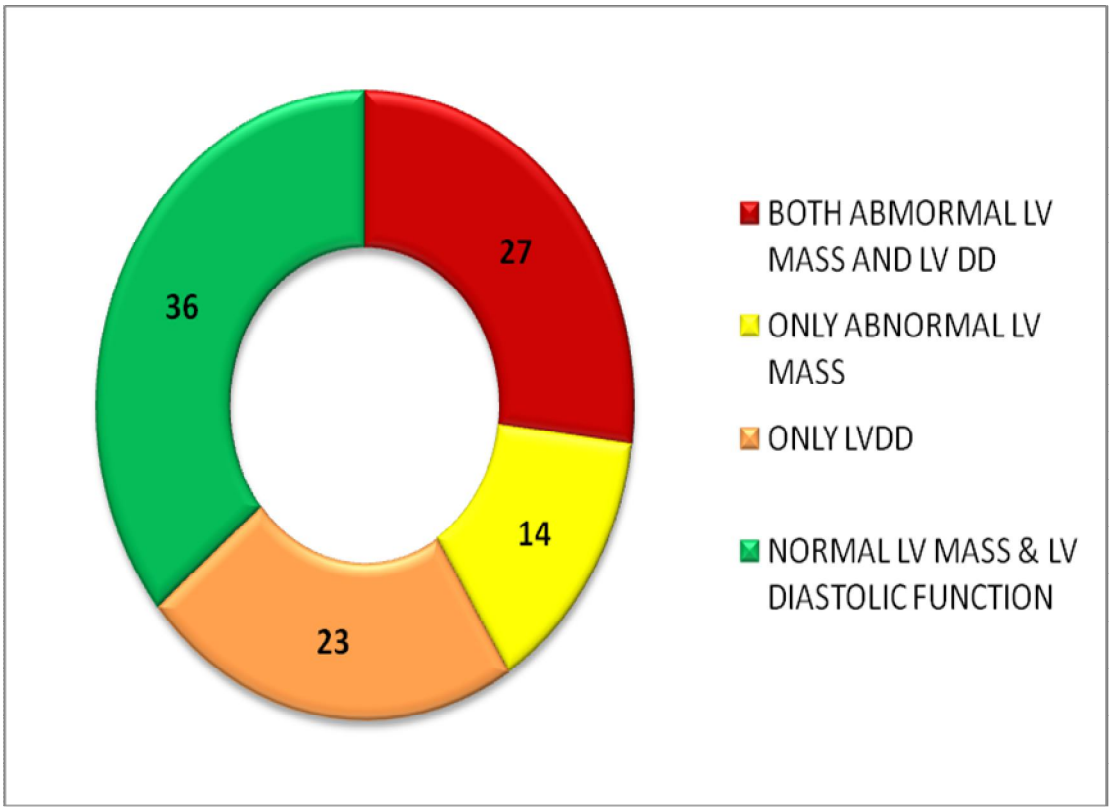

DURATION OF HYPERTENSION
70
20
10
70
60
50
40
30
20
10
0
< 5 YEARS
5-10 YEARS
11-15 YEARS


CONTROL OF HYPERTENSION
26
74
CONTROLLED
INADEQUATE CONTROL

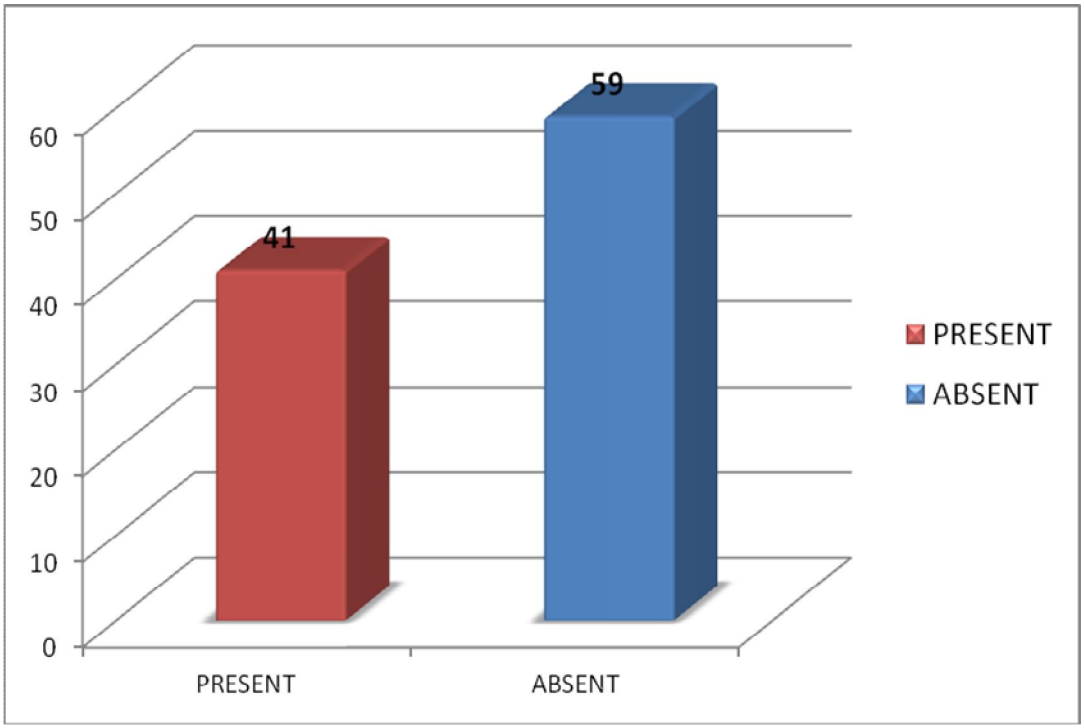

PERCENTAGE OF SMOKERS
19
81
NON - SMOKERS
SMOKERS


BODY MASS INDEX
Normal
26%
Overweight
16%
Obesity
58%

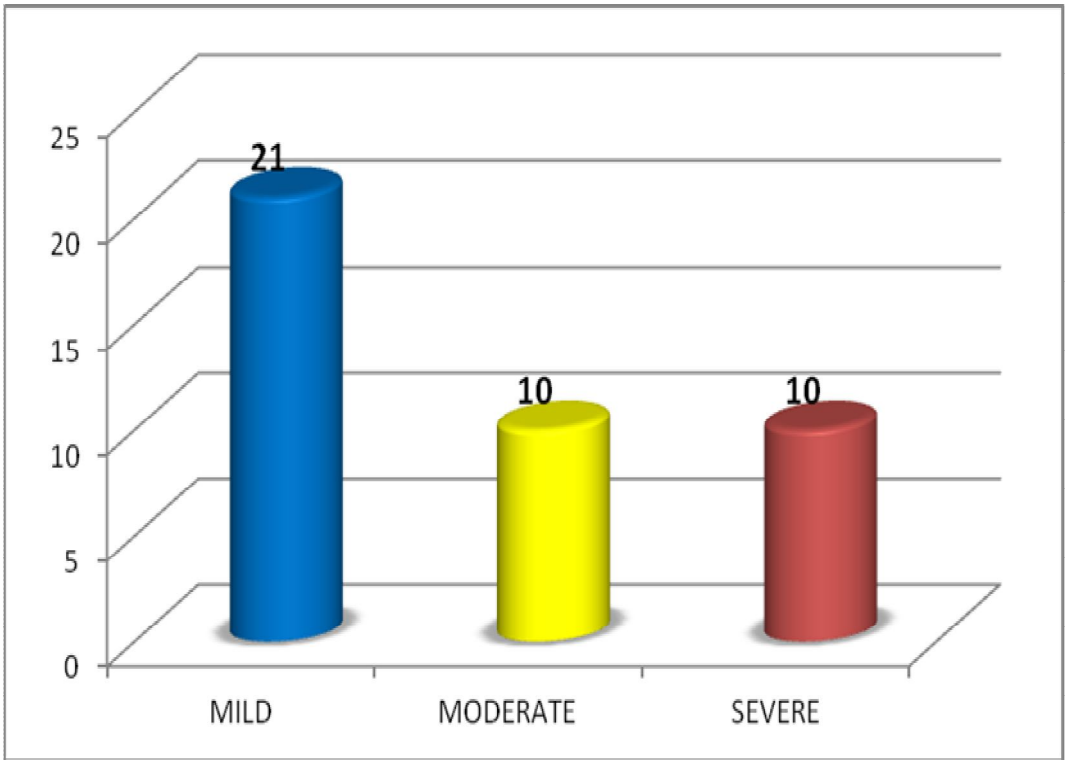
LV MASS AND LVDD



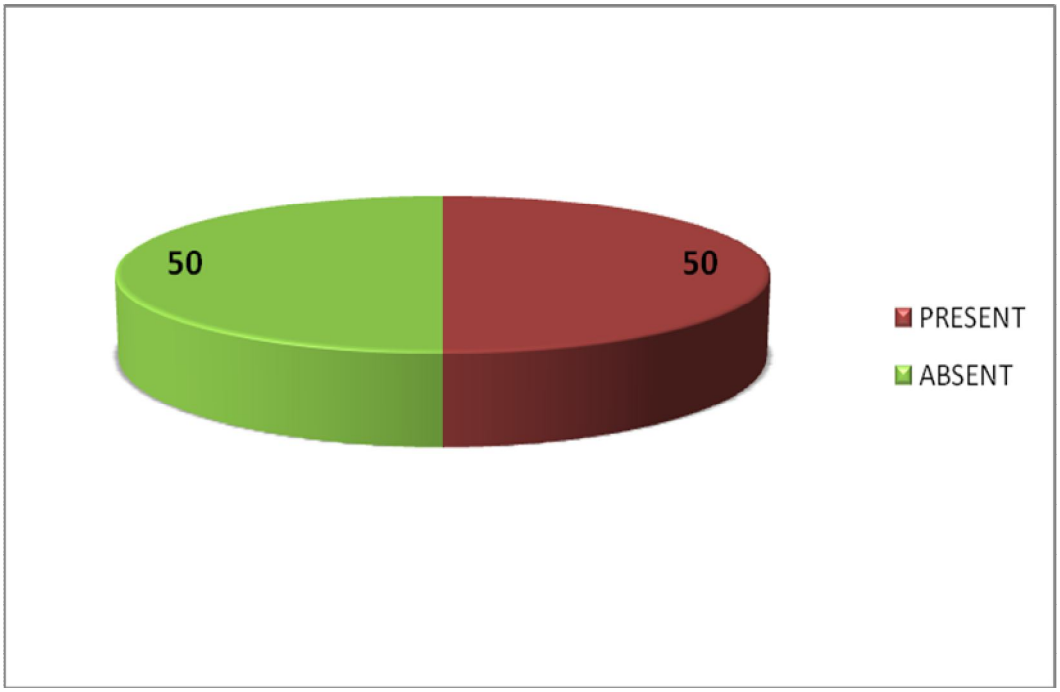
ABNORMAL LV MASS AMONG HYPERTENSIVES



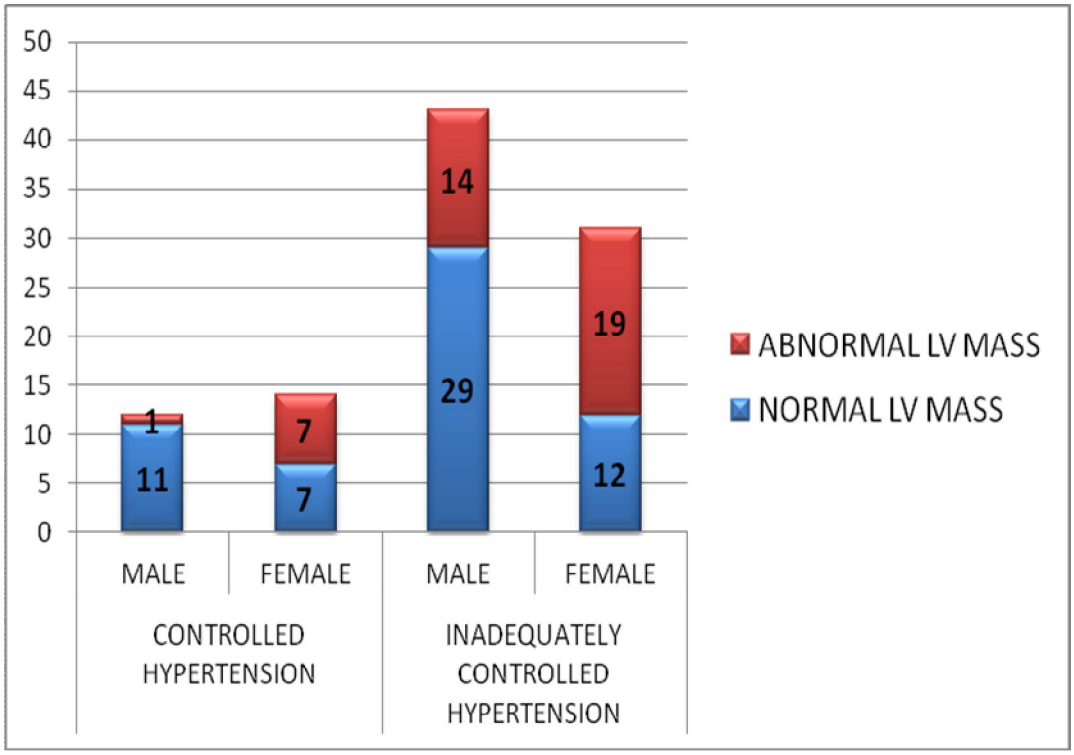
GRADING OF ABNORMAL LV MASS AMONG HYPERTENSIVES



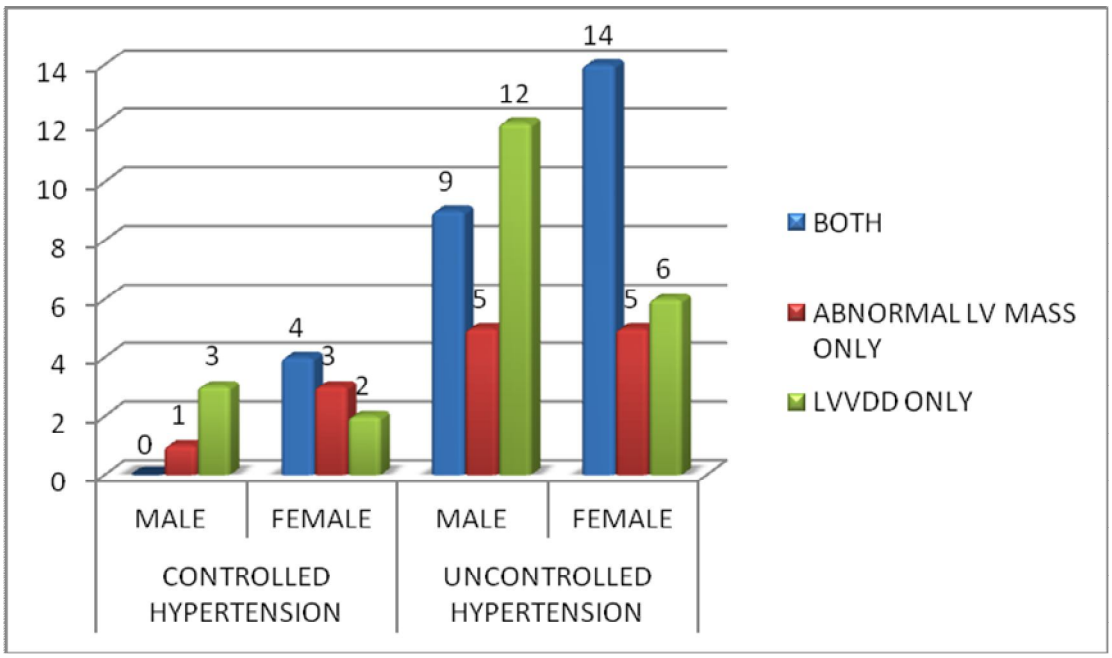
LV DIASTOLIC DYSFUNCTION AMONG HYPERTENSIVES



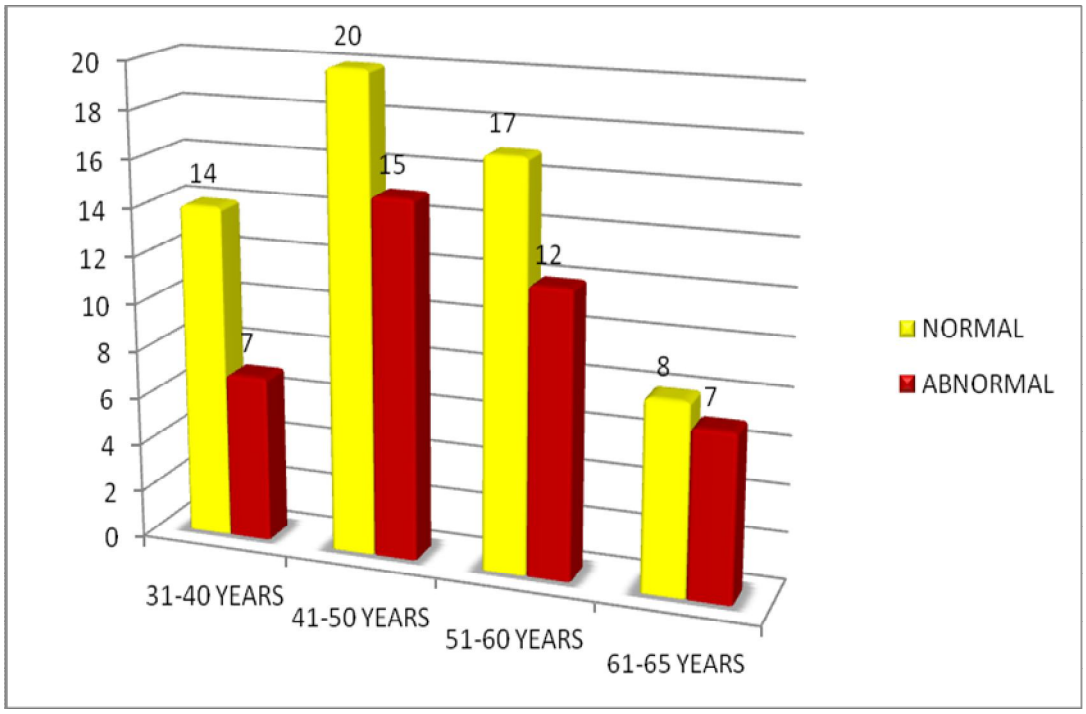
SEVERITY OF HYPERTENSION AND LV MASS AND SEX DISTRIBUTION



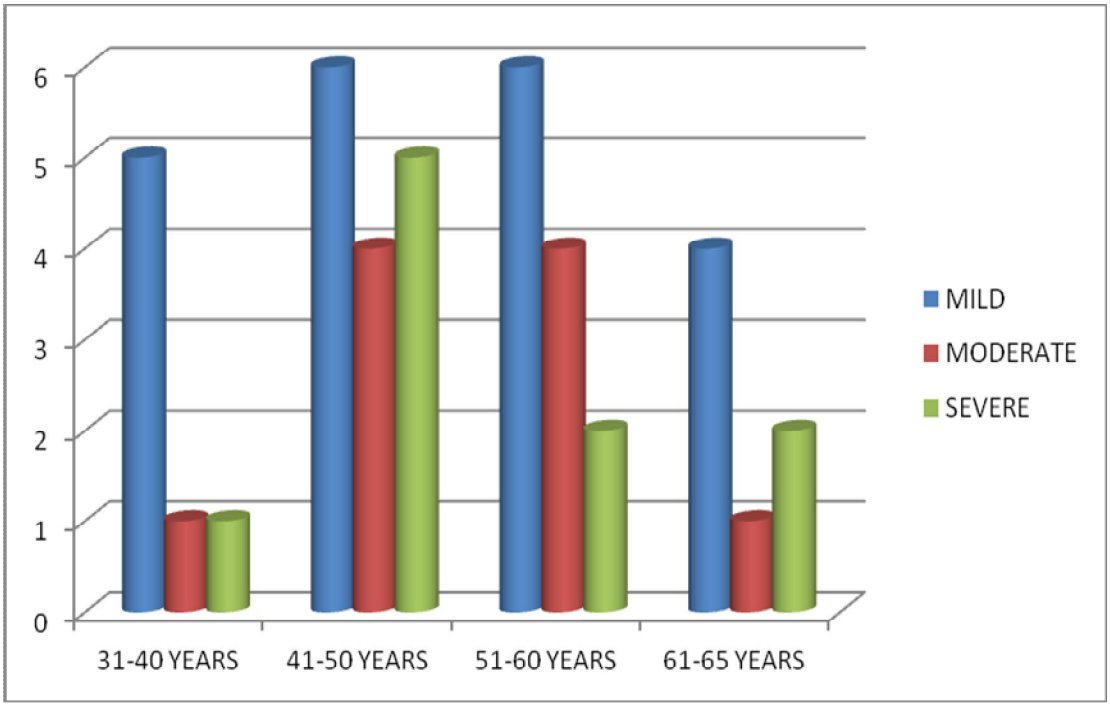
SEVERITY OF HYPERTENSION AND LV MASS AND LV DIASTOLIC FUNCTION AND SEX DISTRIBUTION



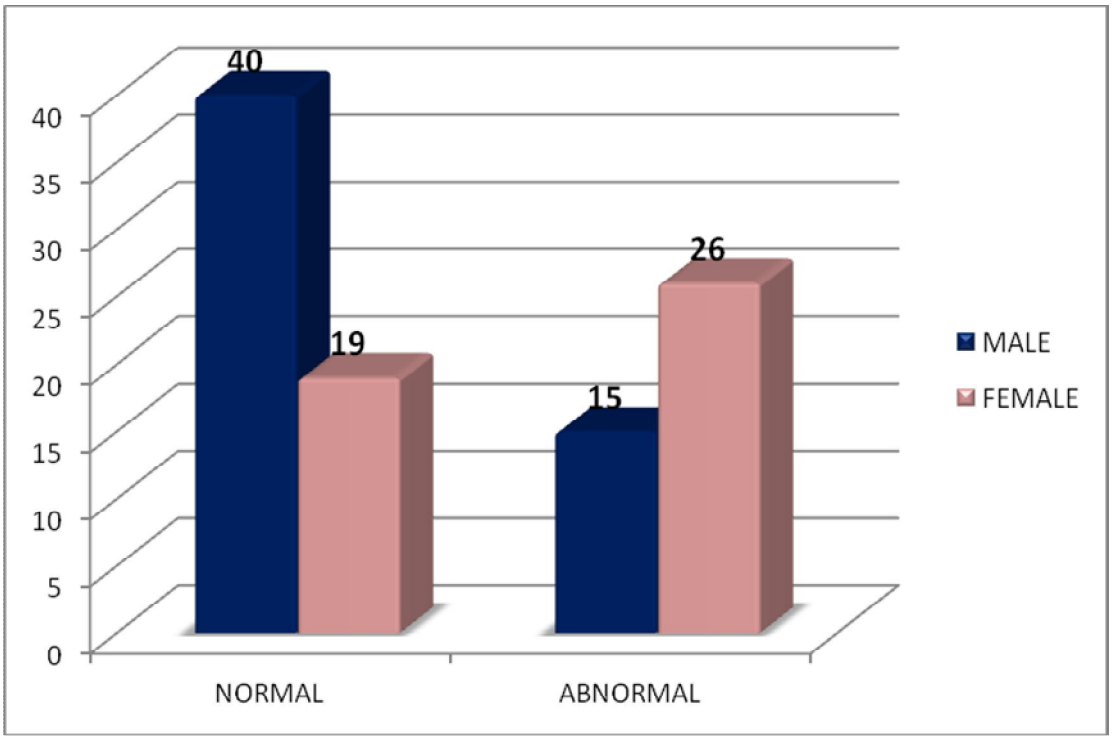
LV MASS AND AGE



LV MASS GRADING AND AGE



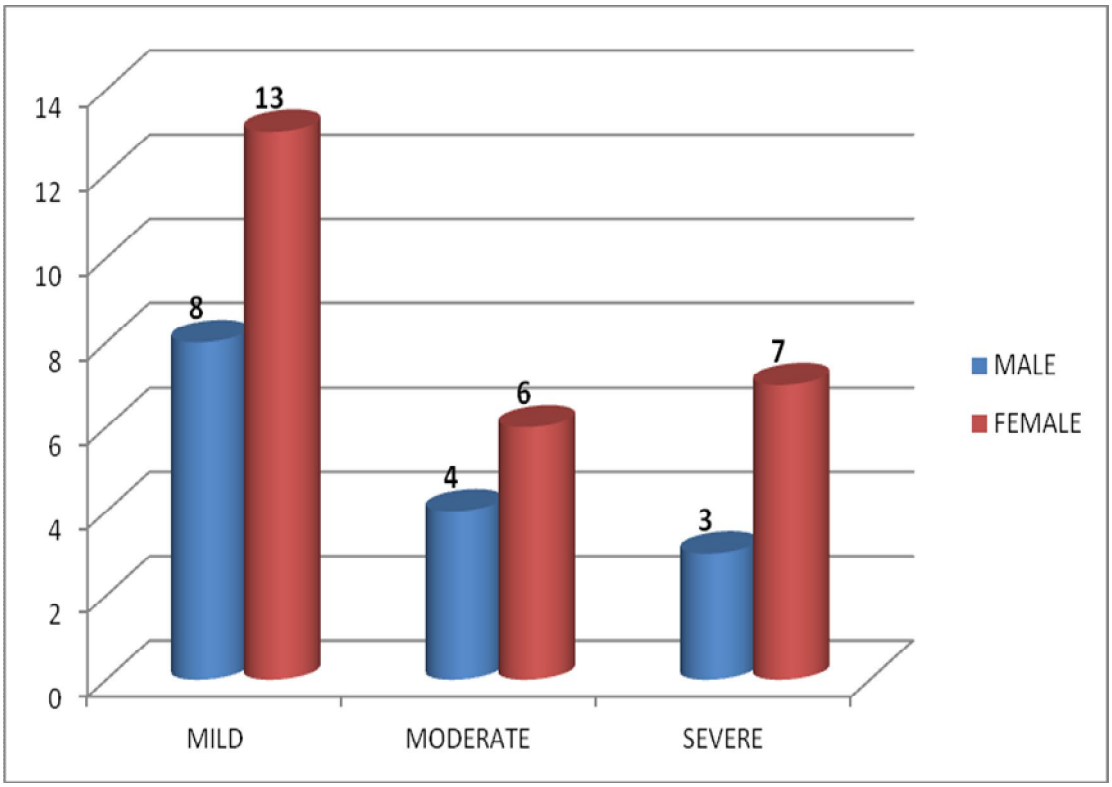
LV MASS AND SEX



Chi – Square test

	value	df	Asymp.Sig
Pearson Chi-square	9.521	1	.002

LV MASS GRADING AND SEX



Chi – Square test

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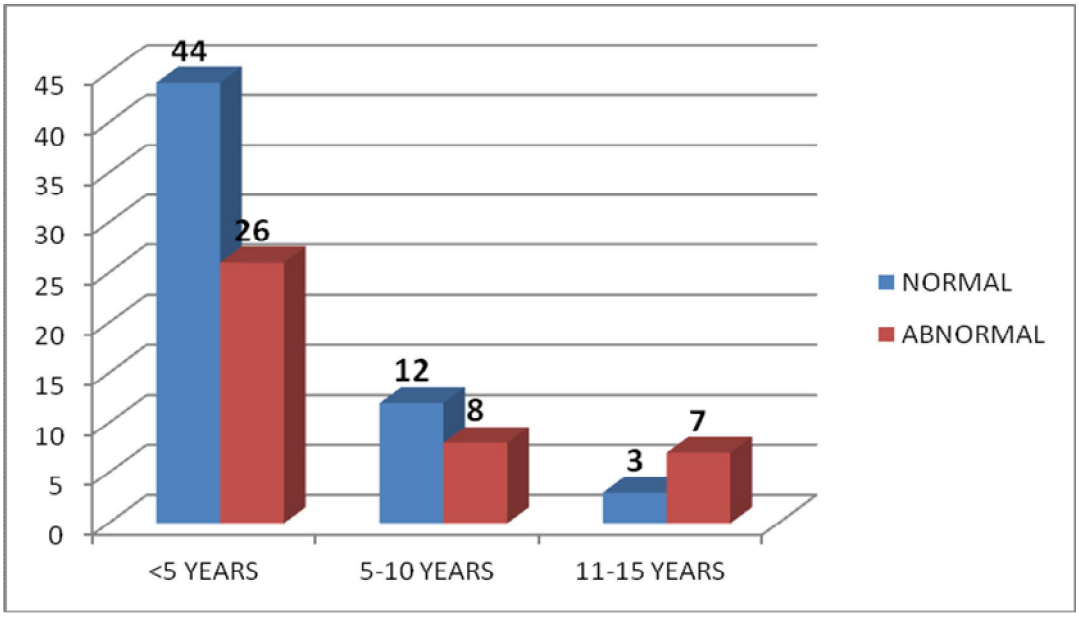
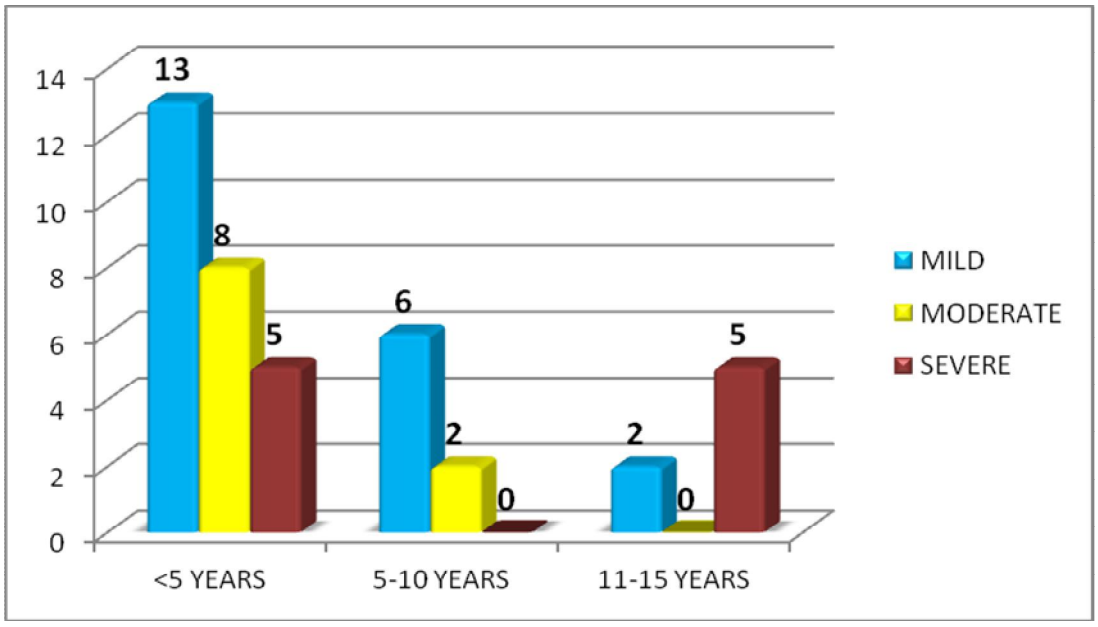
Pearson Chi-square	9.7631	3	.021

MASS AND DURATION OF HYPERTENSION



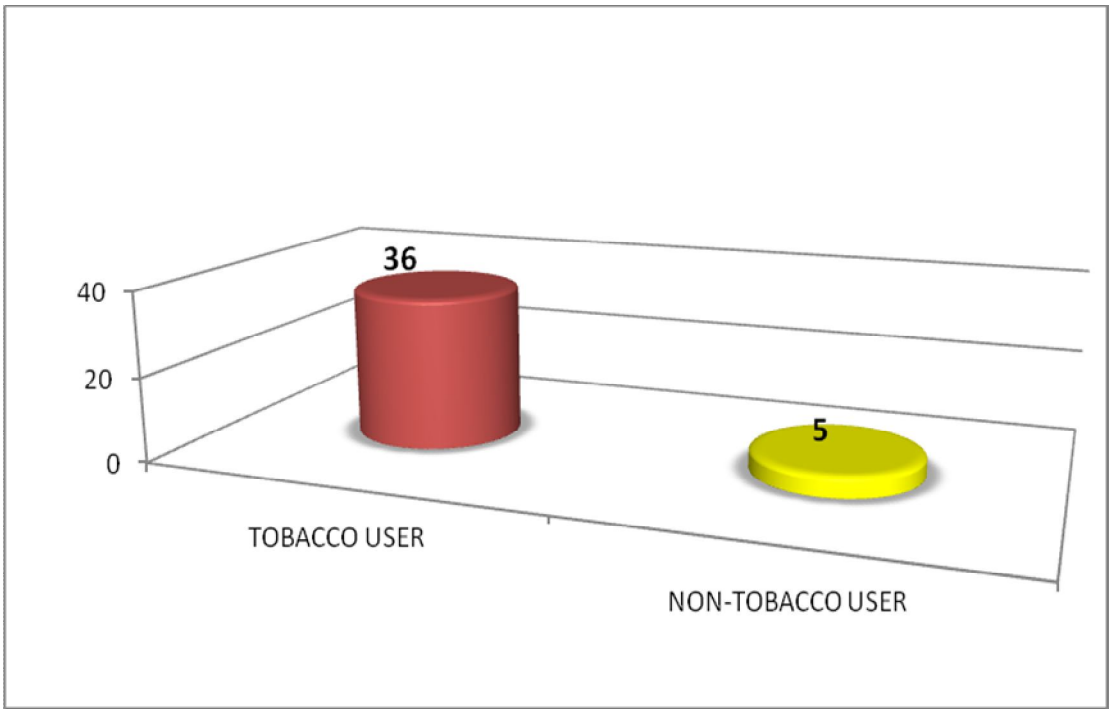
LV MASS GRADING AND DURATION OF HYPERTENSION



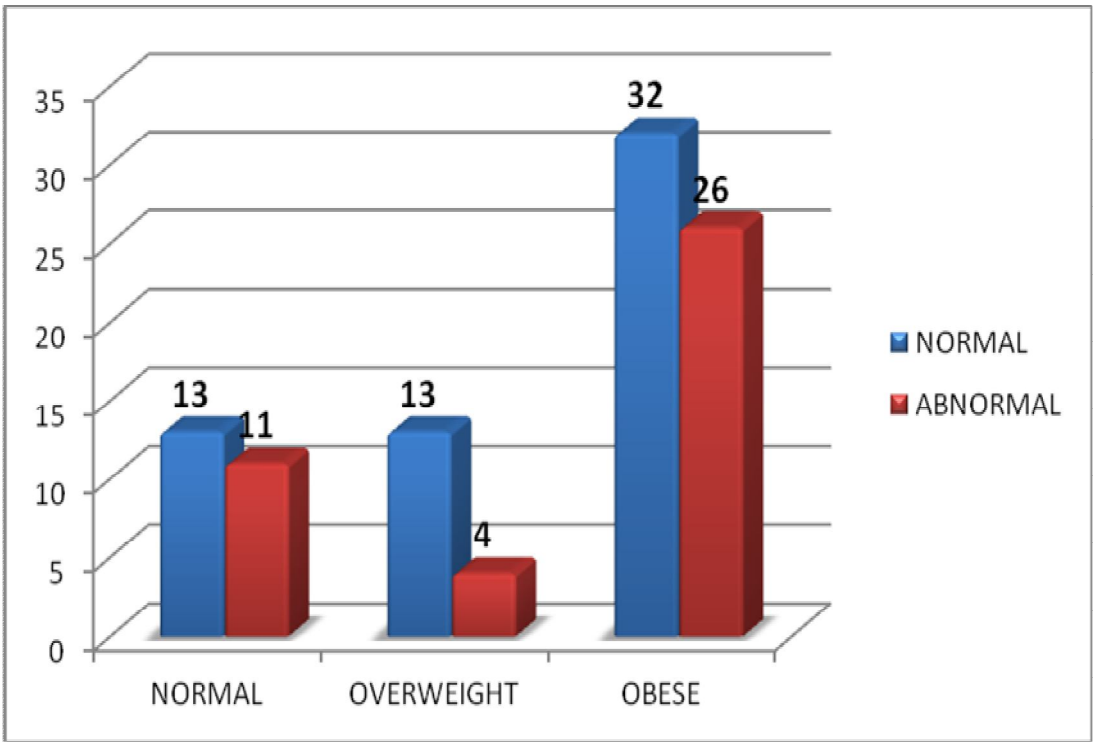
Chi – Square test

	value	df	Asymp.Sig
Pearson Chi-square	22.292	6	.001

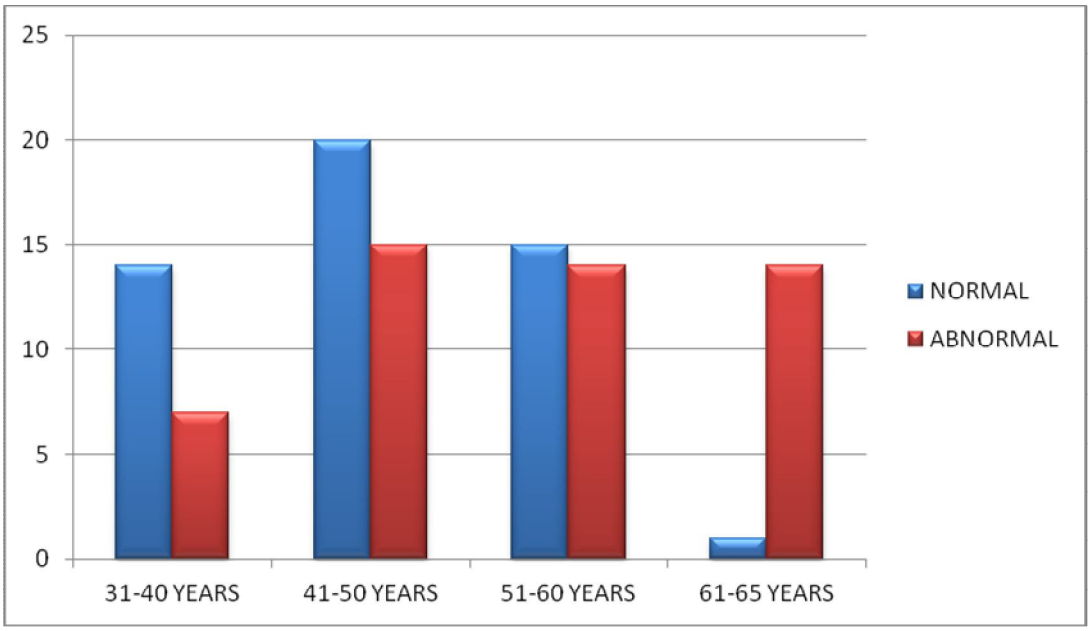
LV MASS AND TOBACCO USE



LV MASS AND BMI



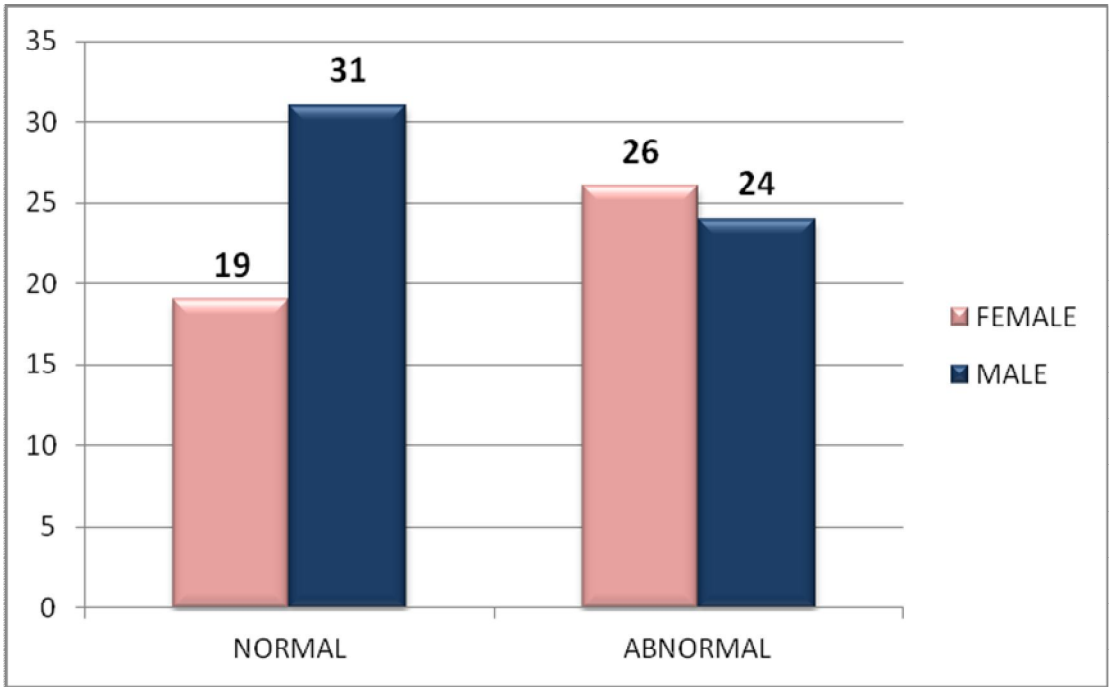
LV DIASTOLIC FUNCTION AND AGE



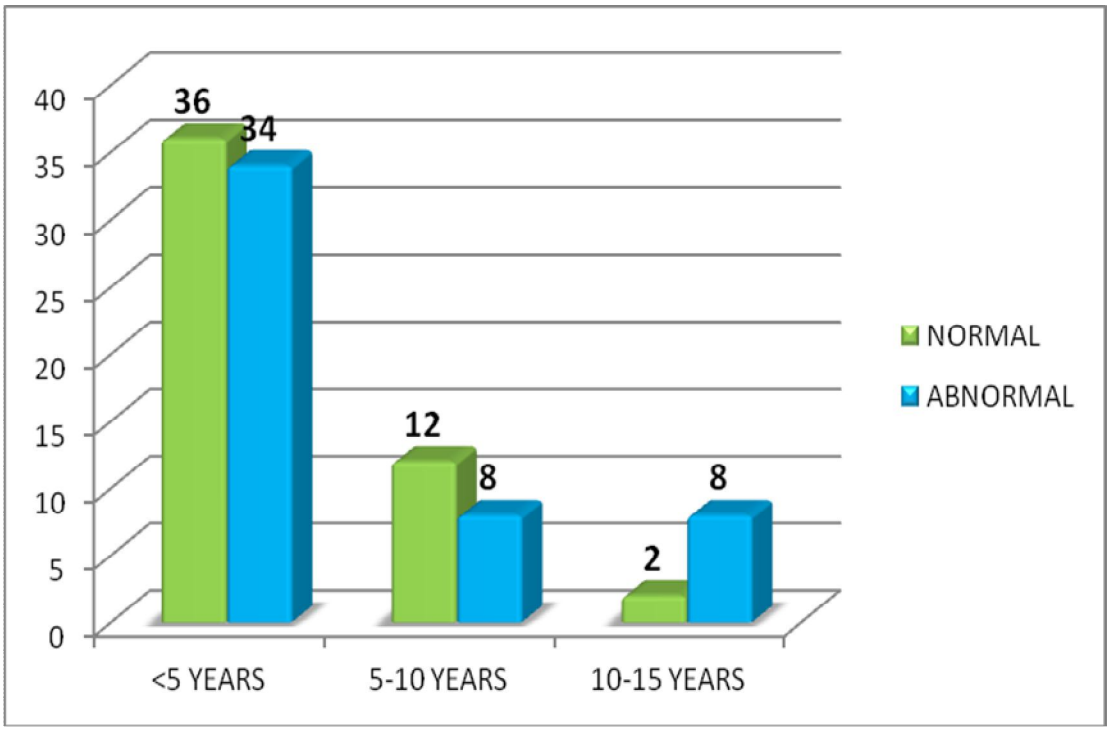
Chi – Square test

	value	df	Asymp.Sig
Pearson Chi-square	14.349	3	.002

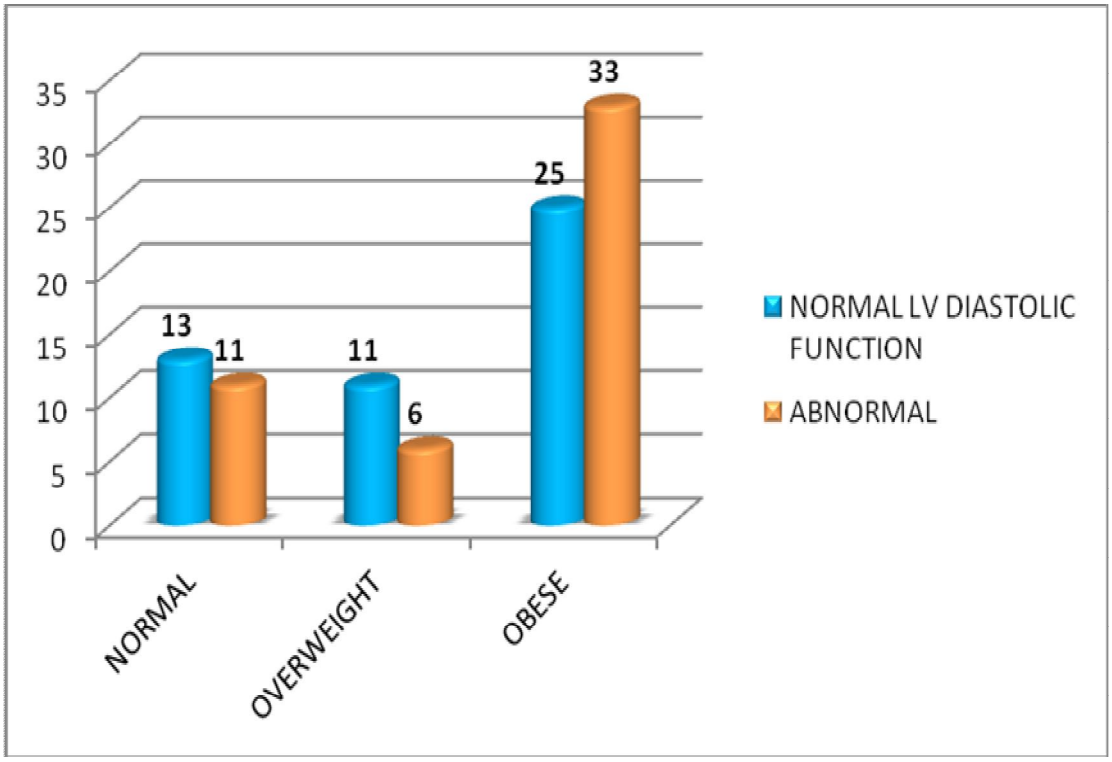
LV DIASTOLIC FUNCTION AND SEX



LV DIASTOLIC FUNCTION AND DURATION OF HYPERTENSION



LV DIASTOLIC FUNCTION AND BMI



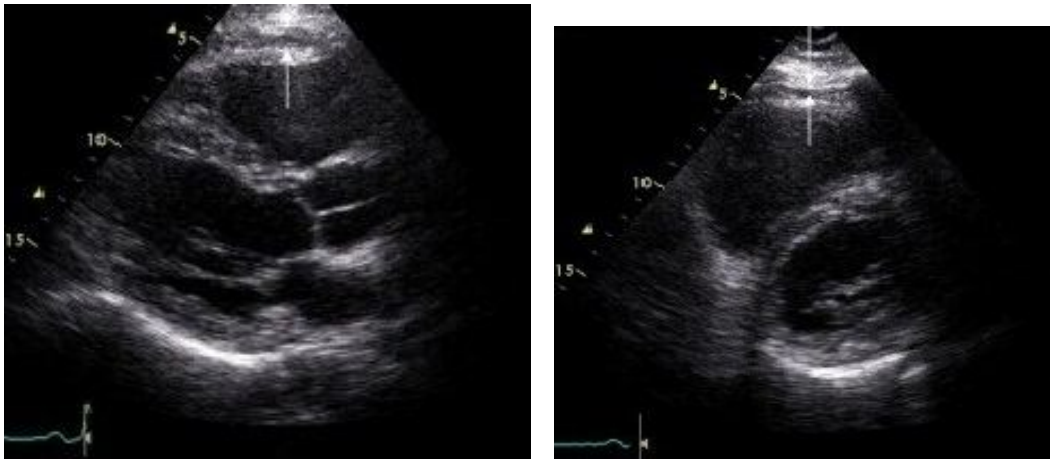


FIG 5.1: *Left ventricular hypertrophy in parasternal long and short axis view*

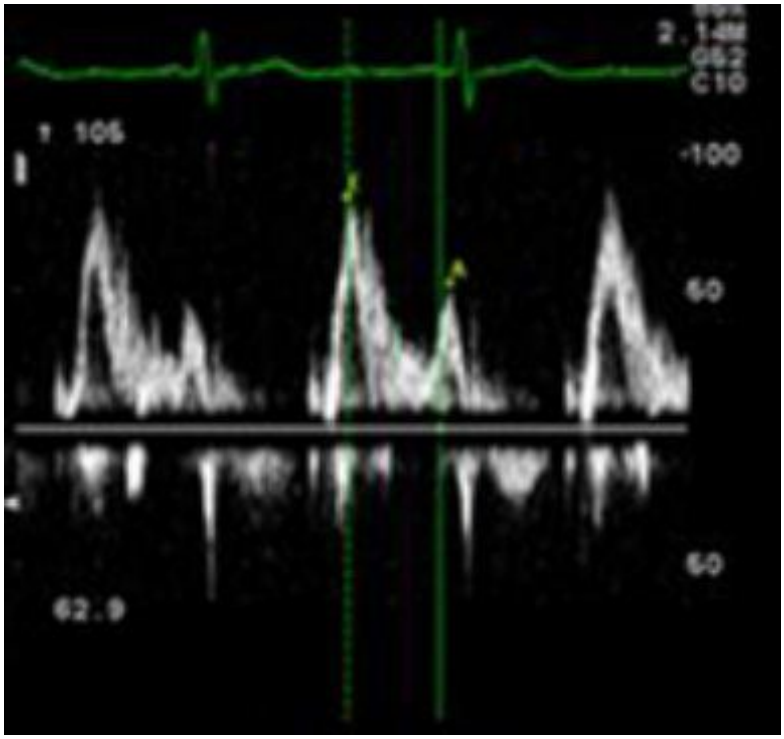


FIG 5.2: *M-mode view showing normal LV diastolic function*

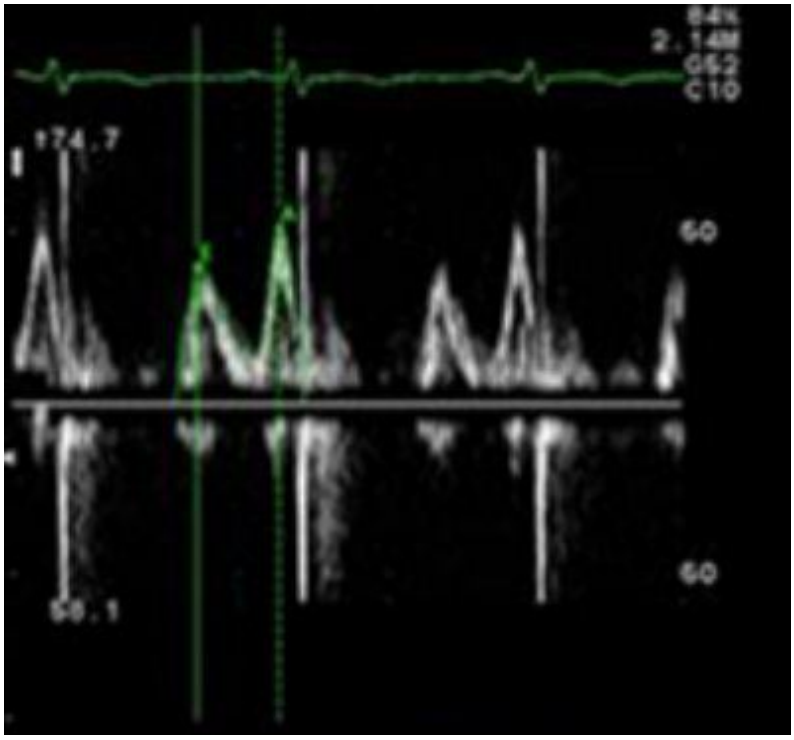


FIG5.3: *M-mode view showing Grade I LVDD*

(E velocity is decreased, A velocity is increased , DT prolonged)

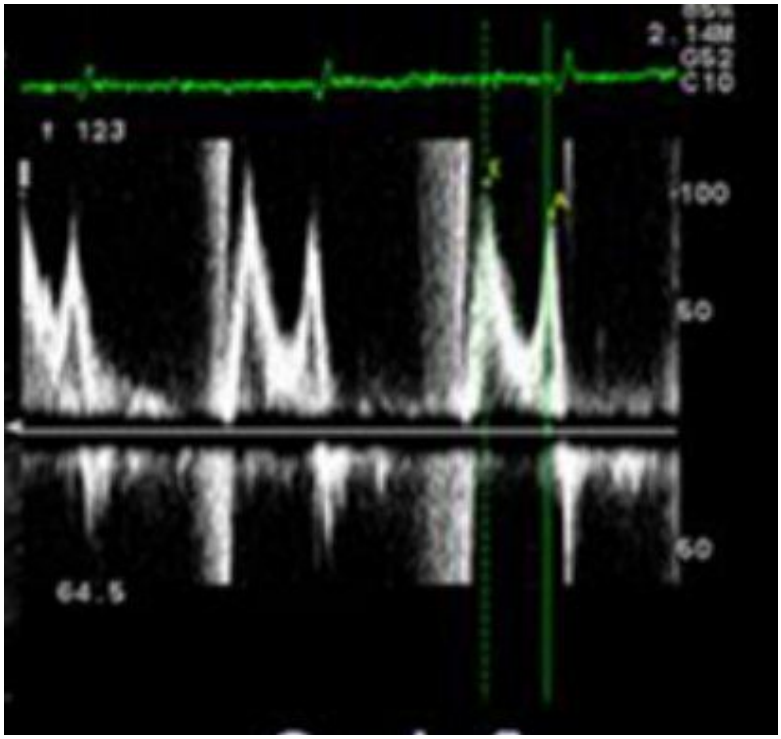


FIG 5.4: *M-mode view showing Grade II LVDD (pseudonormalization)*

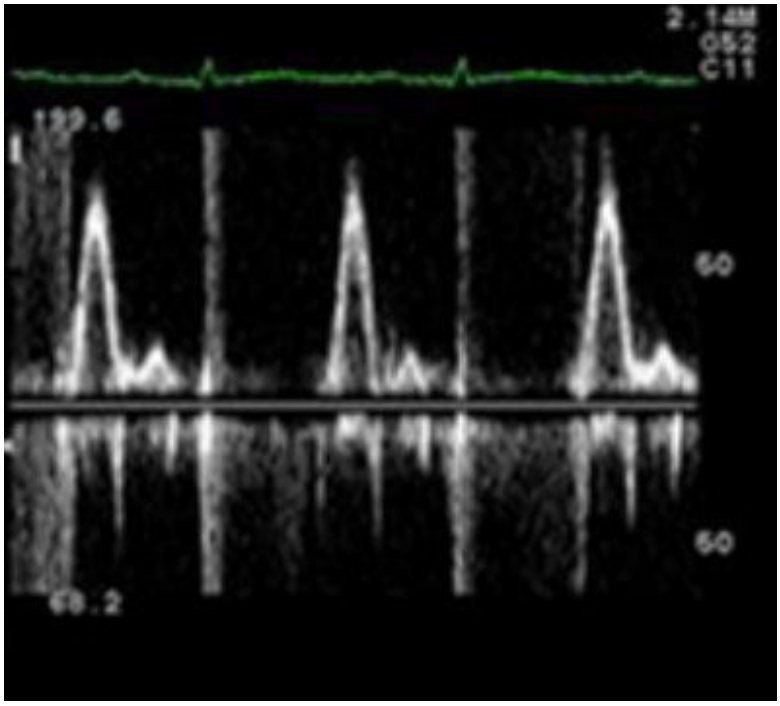


FIG 5.5: *M-mode view showing Grade III LVDD*
(E velocity markedly increased and A velocity markedly decreased, DT shortened)

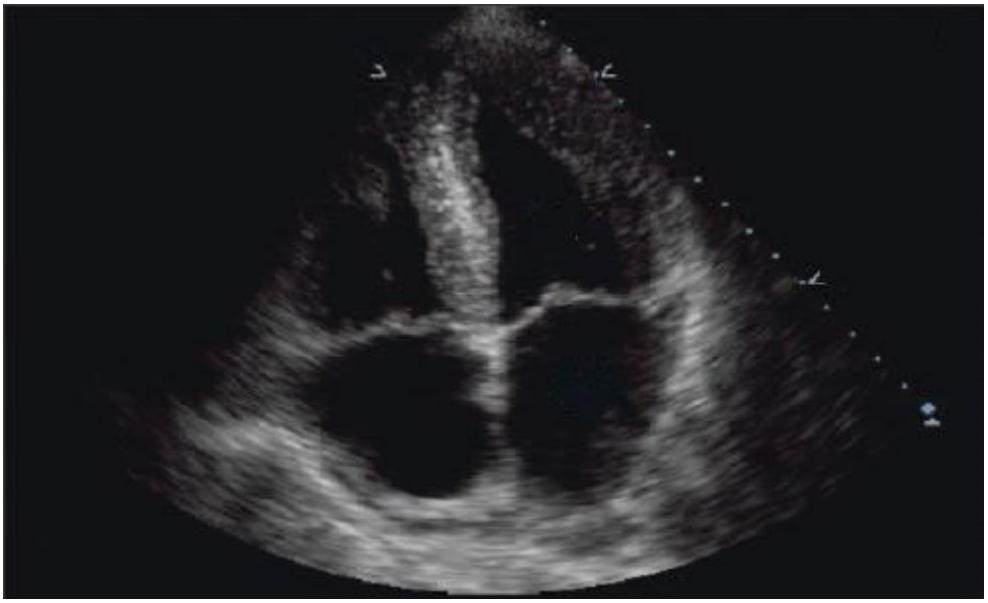


FIG 5.6: *Apical four chamber view showing Left atrial enlargemet along with LVH*

DISCUSSION

6. DISCUSSION

In our study, there were almost equal numbers of male and female participants. 35% of patients were in the age group of 41 to 50 years followed by 29% of patients in the age group of 51-60 years. 70 % of the patients had duration of hypertension for less than 5 years.

It is important to note that 74% of patients had inadequate control of Blood Pressure (defined as SBP ≥140 mmHg and/or DBP ≥90 mmHg at the time of echocardiographic examination as per JNC7 guidelines for goal of therapy). The most important causes for inadequately controlled BP are inappropriate drug doses or drug combinations and improper lifestyle.

19% of the study population were tobacco users of which 16% were females. According to Mishra et al consensus statement for classification of obesity by BMI, 58% were obese and 16% were overweight. This is to emphasize the fact that Hypertension is a feature of metabolic syndrome.

There was only 1 patient with aortic root dilatation and 7 patients had echocardiographically enlarged left atrium.

64% of patient had echocardiographic abnormality of left ventricle. 50% of sample group had LV diastolic dysfunction while 41% had abnormal LV mass (defined as value > 163g in females and >224g in males). 27% of hypertensives had both abnormal LV mass and LV diastolic dysfunction. Approximately 51% had mildly abnormal LV mass and approximately 24% each had moderately and severely abnormal LV mass. Echocardiogram was found to be more sensitive in detectind abnormal LV mass as anticipated

It was noted from our observation that with increasing age , there was proportional increase in LV mass (from 33% in 31-40 age group to 47% in 61-65 age group) which is along the expected lines.

One of the important implications of our study was the influence of sex over LV mass. 27% of male hypertensives had abnormal LV mass while it was 58% for female hypertensives. Overall 63% of those with abnormal LV mass were female hypertensives and it was statistically very significant.

Even when the duration of hypertension was less than 5 years, 37% of hypertensives had abnormal LV mass of which 50% was mild. When the duration of hypertension was more than 10 years, 70% of patients had abnormal LV mass of which statistically significant 71% had severely abnormal LV mass.

90% of those who had abnormal LV mass were tobacco users and 58% of patients with inadequately controlled hypertension were males. 61% of females with inadequate control of hypertension had abnormal LV mass.

With advancing age, we observed proportional increase in the incidence of diastolic dysfunction from 33% in 31-40 age group to 48% in 50-60 age group to 93% in 61-65 age group. We found statistically significant diastolic dysfunction with increasing age that could probably be explained by age related myocardial stiffening.

Both males and females almost equally had LV diastolic dysfunction and as anticipated LV diastolic dysfunction increased with increased in the duration of hypertension.

57% of obese hypertensives had LV diastolic dysfunction and 45% of obese hypertensives had abnormal LV mass. The independent role of obesity causing diastolic dysfunction has to be examined further with equal recruitment of obese and non obese individuals.

LIMITATIONS:

The following was found to be the limitations of the study

- The sample size was small and further studies with larger number of people in multi centers representing different ethnic groups have to be done to verify the results.
- Majority of participants were obese, had inadequate BP control, non-tobacco user and equal proportion on BMI, BP control and tobacco and non tobacco use should have been tested.
- Tissue Doppler Imaging which is ideal for evaluating LV diastolic dysfunction should have used

CONCLUSION

7. CONCLUSION

- Half of the study group – asymptomatic hypertensives- had evidence of LV diastolic dysfunction. This emphasizes the role of screening asymptomatic hypertensives for LV diastolic function, as diastolic dysfunction is a fore runner of heart failure in future

- According to our study, Female sex is a risk factor for developing abnormal LV mass which has to be clarified with further larger sample size recruitment.

- Even a short duration of hypertension i.e., less than 5 years results in abnormal LV mass in one third of patients. Hence, earlier achievement of target BP helps in preventing the progression of end organ damage.

- Most of those who had abnormal LV mass were tobacco users. Therefore, influence of tobacco over LV mass has to further scrutinized

- Two-thirds of the study population were obese and two-thirds of them had LV diastolic dysfunction. This underlines the importance of obesity as a risk factor for hypertension and heart failure.

- Importance of life style education and compliance of drugs is implied by the fact that three-fourths of hypertensives had uncontrolled BP

- The study reiterates the fact that there is a proportional age related increased incidence of LV diastolic dysfunction and abnormal LV mass

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BIBLIOGRAPHY

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ANNEXURES

ANNEXURES
DATA COLLECTION SHEET

NAME :	AGE /SEX :		OP. NO
ADDRESS:	OCCUPATION:		
HYPERTENSION DETAILS			
DURATION	ANTI HYPERTENSIVE DRUGS		
TOBACCO USE: YES / NO	Height(cm):	Weight (Kg):	BP (mmHg):
Anti-Hypertensive Drug details:	Blood Glucose (mg/dl)	Blood urea (mm/dl)	Serum creatinine (mg/dl)
ECG	Chest X-Ray		

ECHOCARDIOGRAPHY REPORT

LVIDd	LVIDS	EF	Left Atrial Area
IVd	IVs	Aortic root Diameter	Flow abnormalities
PWd	PWs	Left Atrial diameter	RWMA : YES/NO
MV (E)	MV (A)	E/A	MV DecT
REMARKS			

PARTICIPANT CONSENT FORM

Participant's name: Address:

Title of the project: ECHOCARDIOGRAPHIC CHANGES ASSOCIATED WITH SYSTEMIC HYPERTENSION IN HYPERTENSIVE PATIENTS IN GOVERNMENT ROYAPETTAH HOSPITAL, CHENNAI – AN OBSERVATIONAL STUDY

The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). I have been given an information sheet giving details of the study. I fully consent to participate in the above study.

Signature of the participant: _____

Date: _____

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No.6206/ME-1/Ethics/2012 Dt:05.07.2012.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on Echocardiographic changes associated with systemic hypertension in hypertensive patients in Govt. Royapettah Hospital, Chennai – An Observational Study submitted by Dr.S.Sridhar, MD (General Medicine), PG Student, Govt. Royapettah Hospital, Chennai

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

27/12/12

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College, Chennai

DEAN,
KILPAUK MEDICAL COLLEGE
CHENNAI-600 010.

27/12

GOVT. KILPAUK MEDICAL COLLEGE
27 DEC 2012
Kilpauk, Chennai-10.

MASTER CHART

S.NO	PATIENT'S NAME	AGE	SEX	DURATION (YEARS)	TOBACCO	BMI	BMI GRADING	BP	SEVERITY	AR(cm)	AR /M2	AORTIC ROOT DILATATION	LA(cm)	LVIDd(cm)	LVIDs(cm)	IVd(cm)	IVs(cm)	PWd(cm)	PWs(cm)	LV MASS(g)	LV MASS	LV MASS GRADING	EF(%)	LAA(cm2)	LAE	MV E	MV A	MV E/A	MV DecT(MS)	LV DIASTOLIC FUNCTION
1	ASMATHUNISHA	62	F	6	NO	32.05	OBESE	130/80	ONTROLLE	3.00	1.23	NO	2.60	4.10	2.40	1.00	1.40	1.50	1.70	182.00	ABNORMAL	MILD	72	13.20	NORMAL	0.76	1.00	0.75	288	ABNORMAL
2	LAKSHMI	38	F	2	NO	27.39	OBESE	130/90	CONTROLL	2.61	1.19	NO	2.74	4.30	2.90	1.20	2.70	1.40	1.60	208.00	ABNORMAL	MODERATE	80	14.22	NORMAL	0.68	0.84	0.81	268	ABNORMAL
3	SELVARAJ	64	M	12	NO	21.87	NORMAL	150/100	CONTROLL	3.11	1.94	NO	3.10	5.10	2.80	1.50	2.10	1.50	1.70	332.00	ABNORMAL	SEVERE	69	15.91	NORMAL	1.25	1.33	0.64	290	ABNORMAL
4	SASIKUMAR	35	M	0.6	NO	19.14	NORMAL	150/90	CONTROLL	3.23	1.26	NO	2.80	4.90	2.80	1.40	2.80	1.60	1.80	313.00	ABNORMAL	SEVERE	74	16.90	NORMAL	1.02	0.54	1.88	154	ABNORMAL
5	BALASUNDARAM	49	M	1	YES	28.05	OBESE	120/80	ONTROLLE	3.30	1.24	NO	2.70	4.50	2.70	1.20	1.80	1.00	1.30	175.00	NORMAL	NORMAL	71	15.29	NORMAL	0.78	1.09	0.72	264	ABNORMAL
6	INDRA	43	F	5	NO	29.77	OBESE	130/80	ONTROLLE	3.41	1.42	NO	3.00	5.00	3.10	1.10	1.50	0.90	1.20	170.00	ABNORMAL	MILD	68	18.00	NORMAL	1.18	0.80	1.47	138	NORMAL
7	AMBUJAM	56	F	2	NO	25.91	OBESE	150/90	CONTROLL	2.75	1.27	NO	2.96	4.50	3.00	1.10	1.40	0.90	1.10	153.00	NORMAL	NORMAL	61	16.70	NORMAL	0.90	0.82	1.10	120	NORMAL
8	KAVERI	65	F	1	YES	28.69	OBESE	130/90	CONTROLL	3.82	1.65	NO	2.90	4.60	2.80	1.00	1.50	0.90	1.20	148.00	NORMAL	NORMAL	65	14.30	NORMAL	0.90	1.16	0.78	288	ABNORMAL
9	KHALEEL	63	M	12	NO	29.13	OBESE	140/96	CONTROLL	3.31	1.38	NO	2.70	4.90	3.30	1.20	1.60	0.80	1.10	176.00	NORMAL	NORMAL	61	17.70	NORMAL	0.71	0.91	0.78	290	ABNORMAL
10	PARTHASARATHY	63	M	14	NO	34.29	OBESE	140/90	CONTROLL	3.30	1.26	NO	2.80	5.30	3.60	1.10	1.70	1.10	1.40	228.00	ABNORMAL	MILD	61	19.50	NORMAL	0.60	1.06	0.57	292	ABNORMAL
11	SURESHBABU	33	M	2	YES	28.89	OBESE	138/84	ONTROLLE	3.00	1.03	NO	3.50	5.10	3.10	0.90	1.30	0.80	1.20	152.00	NORMAL	NORMAL	68	15.35	NORMAL	1.33	1.01	1.31	132	NORMAL
12	GOPAL	43	M	3	NO	25.09	OBESE	120/90	CONTROLL	3.14	1.12	NO	2.60	4.20	2.00	1.20	1.50	1.20	1.50	178.00	NORMAL	NORMAL	78	17.16	NORMAL	1.02	0.68	1.50	138	NORMAL
13	JAISANKAR	43	M	6	NO	30.48	OBESE	130/80	ONTROLLE	3.40	1.22	NO	2.90	5.10	2.80	1.00	1.50	1.00	1.30	188.00	NORMAL	NORMAL	78	18.00	NORMAL	1.29	1.04	1.24	114	NORMAL
14	BAASHA	52	M	5	NO	28.48	OBESE	130/80	ONTROLLE	3.60	1.42	NO	4.40	5.30	2.80	1.10	1.80	1.00	1.30	214.00	NORMAL	NORMAL	69	24.30	ABNORMAL	0.89	0.68	1.31	130	NORMAL
15	MOHAMMED	65	M	1	NO	25.81	OBESE	136/90	CONTROLL	3.90	1.62	NO	2.70	4.40	2.40	1.20	1.90	1.10	1.30	180.00	NORMAL	NORMAL	72	13.40	NORMAL	0.78	1.22	0.64	288	ABNORMAL
16	SULTAN	63	M	2	NO	26.14	OBESE	160/100	CONTROLL	3.42	1.44	NO	3.20	5.10	2.60	1.20	1.80	0.90	1.20	201.00	NORMAL	NORMAL	79	18.00	NORMAL	0.58	0.88	0.66	288	ABNORMAL
17	SHAKEELA BANU	39	F	0.6	NO	32.47	OBESE	130/90	CONTROLL	3.30	1.37	NO	3.00	5.10	2.40	0.90	1.40	0.80	1.10	152.00	NORMAL	NORMAL	66	18.00	NORMAL	1.03	0.81	1.27	120	NORMAL
18	ANNAMMA	46	F	0.8	NO	34.13	OBESE	142/90	CONTROLL	2.92	1.22	NO	3.00	4.60	2.70	0.90	1.40	0.80	1.10	128.00	NORMAL	NORMAL	72	17.00	NORMAL	0.99	0.94	1.05	150	NORMAL
19	CHOODAMANI	38	F	1	NO	26.30	OBESE	130/80	ONTROLLE	3.00	1.23	NO	3.00	5.10	3.10	1.00	1.50	0.80	1.10	163.00	ABNORMAL	MILD	68	15.60	NORMAL	1.04	0.71	1.47	138	NORMAL
20	BHUVANESHWARI	44	F	0.5	NO	27.05	OBESE	140/80	ONTROLLE	2.80	1.17	NO	3.80	4.80	2.70	1.10	1.70	0.90	1.20	170.00	ABNORMAL	MILD	76	22.00	ABNORMAL	1.03	0.51	2.04	156	ABNORMAL
21	CHANDRAKALA	31	F	1	NO	19.22	NORMAL	150/90	CONTROLL	2.63	1.05	NO	2.60	4.30	2.70	0.90	1.50	0.80	1.20	114.00	NORMAL	NORMAL	67	15.80	NORMAL	1.28	1.03	1.24	108	NORMAL
22	TAMILSELVI	43	F	12	NO	21.63	NORMAL	160/100	CONTROLL	3.11	1.24	NO	3.10	4.70	2.60	0.90	1.60	0.80	1.10	251.00	ABNORMAL	SEVERE	75	17.00	NORMAL	0.90	0.63	1.42	114	NORMAL
23	LAKSHMI	50	F	2	NO	30.41	OBESE	130/90	CONTROLL	3.10	1.27	NO	3.10	4.60	2.70	1.10	1.90	1.00	1.40	170.00	ABNORMAL	MILD	72	21.40	ABNORMAL	0.99	1.23	0.80	256	ABNORMAL
24	CHARLES	37	M	4	NO	19.14	NORMAL	180/98	CONTROLL	3.00	1.17	NO	3.20	4.60	3.00	1.00	1.40	1.00	1.40	159.00	NORMAL	NORMAL	70	17.00	NORMAL	0.85	0.78	1.10	78	NORMAL
25	VIJAY	37	M	0.6	NO	23.22	VERWEIGH	170/100	CONTROLL	3.30	1.19	NO	3.40	4.20	2.60	1.00	1.60	1.10	1.40	147.00	NORMAL	NORMAL	70	20.10	NORMAL	0.73	0.55	1.30	96	NORMAL
26	MALLIKA	45	F	1	NO	20.23	NORMAL	140/90	CONTROLL	3.54	1.49	NO	2.60	5.10	2.90	1.20	1.60	1.10	1.40	227.00	ABNORMAL	SEVERE	73	16.30	NORMAL	0.60	0.84	0.81	272	ABNORMAL
27	IQBAL	52	M	3	NO	17.84	NORMAL	130/80	ONTROLLE	3.00	1.11	NO	3.10	4.70	2.80	1.00	1.60	1.10	1.50	164.00	NORMAL	NORMAL	72	15.30	NORMAL	0.72	0.66	1.08	96	NORMAL
28	PRABHAKAR	42	M	2	NO	26.50	OBESE	140/94	CONTROLL	3.60	1.42	NO	3.90	4.50	3.00	1.00	1.60	1.00	1.30	153.00	NORMAL	NORMAL	62	22.00	ABNORMAL	0.80	0.50	1.50	174	NORMAL
29	VALLIKANNU	56	M	5	NO	23.11	VERWEIGH	132/90	CONTROLL	2.90	1.28	NO	2.90	4.10	2.60	0.90	1.90	1.00	1.40	118.00	NORMAL	NORMAL	66	13.70	NORMAL	0.63	0.82	0.78	264	ABNORMAL
30	SELVAM	62	M	5	YES	21.87	NORMAL	150/80	CONTROLL	4.00	1.56	NO	3.30	5.00	3.20	1.00	1.70	1.00	1.40	182.00	NORMAL	NORMAL	67	25.00	ABNORMAL	0.50	0.89	0.60	284	ABNORMAL
31	KARUPPIAH	62	M	0.3	NO	28.07	OBESE	130/90	CONTROLL	3.74	1.23	NO	3.86	5.40	3.30	1.20	1.90	1.20	1.50	264.00	ABNORMAL	MODERATE	72	25.15	ABNORMAL	0.77	0.94	0.82	296	ABNORMAL
32	GOWRI	40	F	1	NO	25.55	OBESE	140/84	CONTROLL	3.10	1.50	NO	2.90	4.60	2.60	0.80	1.50	0.80	1.10	118.00	NORMAL	NORMAL	73	17.00	NORMAL	0.90	0.97	0.79	264	ABNORMAL
33	LAKSHMI	46	F	2	NO	29.97	OBESE	120/80	ONTROLLE	5.40	2.25	YES	3.60	4.80	2.40	0.90	1.20	1.00	1.40	159.00	NORMAL	NORMAL	68	13.20	NORMAL	0.76	1.00	0.76	258	ABNORMAL
34	DEIVAANAI	47	F	3	NO	23.51	VERWEIGH	140/90	CONTROLL	2.90	1.06	NO	3.30	4.70	2.60	1.00	1.50	1.00	1.30	164.00	ABNORMAL	MILD	75	16.00	NORMAL	0.80	0.73	1.20	132	NORMAL
35	PARVATHY	53	F	7	NO	21.78	NORMAL	140/90	CONTROLL	2.80	1.03	NO	2.60	4.60	2.60	0.90	1.30	1.40	1.70	193.00	ABNORMAL	MODERATE	75	14.90	NORMAL	1.03	0.76	1.35	190	NORMAL
36	KUMAR	46	M	2	YES	26.77	OBESE	158/92	CONTROLL	3.33	1.24	NO	3.60	4.80	2.70	1.10	1.60	1.10	1.40	194.00	NORMAL	NORMAL	76	15.40	NORMAL	0.76	0.64	1.20	72	NORMAL
37	THANGAM	38	F	2	NO	20.93	NORMAL	154/90	CONTROLL	2.84	1.21	NO	3.00	4.20	2.60	1.30	1.00	1.10	1.50	178.00	ABNORMAL	MILD	70	17.00	NORMAL	0.61	0.75	0.80	272	ABNORMAL
38	REGINA	43	F	15	NO	22.50	NORMAL	160/90	CONTROLL	2.80	1.21	NO	3.10	4.30	2.80	1.00	1.30	1.00	1.30	142.00	NORMAL	NORMAL	70	16.50	NORMAL	0.88	0.70	1.26	84	NORMAL
39	VENKATESAN	53	M	1	YES	24.91	VERWEIGH	130/80	ONTROLLE	3.00	1.11	NO	3.50	4.50	2.40	1.20	1.60	1.00	1.40	175.00	NORMAL	NORMAL	60	17.00	NORMAL	0.76	0.64	1.20	150	NORMAL

40	DILIP,	31	M	1	YES	24.67	VERWEIGH	138/90	CONTROLL	3.15	1.14	NO	3.27	4.80	3.30	1.40	1.90	1.20	1.50	246.00	ABNORMAI	MILD	60	14.80	NORMAL	0.78	0.52	1.39	102	NORMAL
41	MALLIKA	48	F	3	NO	32.89	OBESE	140/100	CONTROLL	3.90	1.71	NO	3.00	5.00	3.20	1.60	1.90	0.80	1.10	158.00	NORMAL	NORMAL	64	20.10	ABNORMAL	0.61	0.75	0.80	258	ABNORMAL
42	VIJAYALAKSHMI	43	F	12	NO	27.83	OBESE	120/80	ONTROLLE	3.27	1.38	NO	3.75	4.50	3.10	1.70	2.20	1.30	1.60	276.00	ABNORMAI	SEVERE	58	18.77	NORMAL	0.97	0.81	2.20	114	ABNORMAL
43	IRUDHAYARAJ	49	M	3	YES	26.77	OBESE	126/90	CONTROLL	2.85	1.06	NO	3.02	4.80	2.90	1.20	1.50	1.20	1.50	219.00	NORMAL	NORMAL	70	19.80	NORMAL	0.94	0.56	1.67	144	NORMAL
44	SHANKAR	57	M	2	YES	23.42	VERWEIGH	130/90	CONTROLL	3.10	1.21	NO	3.00	4.70	2.80	1.00	1.40	1.20	1.60	187.00	NORMAL	NORMAL	70	16.50	NORMAL	1.14	0.84	1.35	96	NORMAL
45	SARAVANNAN	60	M	4	YES	25.60	OBESE	160/100	CONTROLL	3.40	1.18	NO	3.80	4.80	2.40	1.20	1.80	1.40	1.70	246.00	ABNORMAI	MILD	70	18.25	NORMAL	0.60	1.70	0.75	296	ABNORMAL
46	RAJKUMAR	40	M	0.6	NO	22.49	NORMAL	150/90	CONTROLL	3.10	1.07	NO	3.20	4.90	3.20	1.00	1.40	1.00	1.30	212.00	NORMAL	NORMAL	63	18.50	NORMAL	1.08	0.62	1.75	150	NORMAL
47	ANBUCHEZIAN	41	M	1	YES	28.07	OBESE	176/100	CONTROLL	3.20	0.97	NO	3.60	4.30	2.70	1.10	1.60	1.10	1.40	163.00	NORMAL	NORMAL	68	18.23	NORMAL	1.10	0.68	1.19	96	NORMAL
48	SEELI	57	F	10	YES	29.97	OBESE	160/90	CONTROLL	3.10	1.30	NO	3.30	4.90	2.70	1.10	1.60	1.10	1.50	200.00	ABNORMAI	MODERATE	76	19.20	NORMAL	1.15	0.78	1.47	120	NORMAL
49	MAHESWARI	31	F	0.3	NO	22.22	NORMAL	130/80	ONTROLLE	2.37	1.05	NO	2.50	4.20	2.50	0.80	1.00	0.90	1.30	110.00	NORMAL	NORMAL	73	12.21	NORMAL	0.80	0.73	1.10	96	NORMAL
50	PAAVADAI	51	M	2	NO	21.05	NORMAL	126/84	ONTROLLE	2.67	0.97	NO	2.99	4.40	2.57	0.80	1.20	0.80	1.10	215.00	NORMAL	NORMAL	73	13.04	NORMAL	1.37	0.89	1.54	174	NORMAL
51	CHANDRAKUMAR	63	M	1	NO	27.64	OBESE	120/90	CONTROLL	2.94	1.04	NO	4.52	5.30	5.70	1.10	1.40	1.10	1.50	228.00	ABNORMAI	MILD	68	18.25	NORMAL	0.96	0.85	1.12	150	NORMAL
52	KRISHNAN	55	M	1	NO	23.22	VERWEIGH	140/90	CONTROLL	2.78	1.01	NO	3.30	4.10	2.90	1.10	1.20	0.90	1.20	132.00	NORMAL	NORMAL	61	11.26	NORMAL	0.71	0.92	0.78	256	ABNORMAL
53	KOIKILA	52	F	0.2	NO	22.48	NORMAL	132/94	CONTROLL	3.40	1.42	NO	3.90	4.90	3.30	1.20	1.50	1.20	1.50	389.00	ABNORMAI	SEVERE	60	13.00	NORMAL	0.88	0.96	0.71	278	ABNORMAL
54	RAJASEKAR	55	M	5	NO	23.53	VERWEIGH	150/112	CONTROLL	3.25	1.12	NO	3.48	4.60	3.50	1.10	0.83	0.90	1.30	159.00	NORMAL	NORMAL	70	15.34	NORMAL	0.96	0.71	1.40	144	NORMAL
55	AMARAVATHY	59	F	0.6	NO	21.91	NORMAL	120/80	ONTROLLE	2.41	1.10	NO	2.49	4.30	2.60	0.80	1.20	1.00	1.50	123.00	NORMAL	NORMAL	70	17.63	NORMAL	1.09	0.92	1.18	180	NORMAL
56	MALLIGA	53	F	4	NO	22.51	NORMAL	140/80	ONTROLLE	2.36	1.02	NO	3.49	4.30	2.90	0.80	1.00	0.80	1.20	105.00	NORMAL	NORMAL	61	13.17	NORMAL	1.26	1.08	0.80	288	ABNORMAL
57	VIJAYBABU	45	M	7	NO	26.57	OBESE	140/90	CONTROLL	2.90	1.03	NO	3.14	5.10	3.30	1.00	1.20	1.00	1.30	188.00	NORMAL	NORMAL	64	16.93	NORMAL	1.01	0.76	1.33	168	NORMAL
58	MUNIYAMMAL	55	F	8	NO	29.21	OBESE	180/100	CONTROLL	3.00	1.37	NO	3.65	4.50	2.70	1.10	1.40	1.00	1.30	164.00	ABNORMAI	MILD	69	15.17	NORMAL	0.47	0.93	0.54	294	ABNORMAL
59	ESWARAN	56	M	1	NO	34.57	OBESE	156/88	CONTROLL	3.58	1.33	NO	4.42	5.30	3.00	1.10	1.60	1.20	1.60	242.00	ABNORMAI	MILD	73	17.66	NORMAL	0.89	0.97	0.82	270	ABNORMAL
60	SEKAR	54	M	5	NO	26.35	OBESE	150/90	CONTROLL	2.87	1.08	NO	3.68	4.30	2.80	1.00	1.30	0.90	1.30	133.00	NORMAL	NORMAL	65	15.67	NORMAL	0.98	0.77	1.27	162	NORMAL
61	SRINIVASAN	45	M	0.1	NO	26.93	OBESE	120/80	ONTROLLE	2.73	0.97	NO	3.03	4.00	2.50	1.00	1.20	0.90	1.40	118.00	NORMAL	NORMAL	68	15.66	NORMAL	0.80	0.91	0.80	264	ABNORMAL
62	VINCENT	38	M	3	NO	24.02	VERWEIGH	130/86	ONTROLLE	2.91	1.04	NO	3.19	5.10	3.70	1.10	1.50	1.10	1.40	214.00	NORMAL	NORMAL	55	15.99	NORMAL	0.84	0.67	1.25	228	NORMAL
63	KUMUDHA	42	F	6	NO	21.00	NORMAL	128/90	CONTROLL	2.87	1.31	NO	3.88	5.20	3.60	0.90	1.20	0.90	1.40	101.00	NORMAL	NORMAL	57	14.01	NORMAL	0.82	0.92	0.78	268	ABNORMAL
64	CHANDRAMOHAN	54	M	6	NO	25.28	OBESE	120/70	ONTROLLE	2.56	0.95	NO	2.54	4.30	3.03	0.90	1.30	1.10	1.20	258.00	ABNORMAI	MILD	56	10.47	NORMAL	1.00	0.84	1.18	174	NORMAL
65	KUMAR	46	M	2	NO	27.51	OBESE	130/100	CONTROLL	3.30	1.23	NO	3.60	4.80	2.70	1.10	1.60	1.10	1.30	260.00	ABNORMAI	MODERATE	60	15.40	NORMAL	0.76	0.64	1.20	0.72	NORMAL
66	CHANDRA	50	F	3	YES	28.30	OBESE	142/96	CONTROLL	2.20	1.00	NO	3.77	5.10	2.90	0.90	1.40	0.90	1.30	163.00	ABNORMAI	MILD	75	17.18	NORMAL	0.81	1.06	0.77	268	ABNORMAL
67	JAYARAMAN	50	M	3	NO	26.57	OBESE	158/100	CONTROLL	2.20	0.78	NO	3.77	5.11	2.86	0.91	1.43	0.91	1.30	196.00	NORMAL	NORMAL	75	1.00	NORMAL	1.17	0.98	1.08	242	ABNORMAL
68	SARASWATHY	56	F	3	NO	32.05	OBESE	160/90	CONTROLL	2.77	1.13	NO	3.55	4.90	2.60	0.90	1.00	1.40	1.60	361.00	ABNORMAI	SEVERE	76	13.44	NORMAL	0.58	0.99	0.59	288	ABNORMAL
69	PREMAVATHY	63	F	6	NO	29.90	OBESE	150/90	CONTROLL	2.80	1.35	NO	3.90	4.60	3.40	0.80	1.10	1.00	1.50	138.00	NORMAL	NORMAL	55	14.75	NORMAL	0.86	1.01	0.85	288	ABNORMAL
70	KUMUDHA	37	F	4	NO	24.65	VERWEIGH	138/94	CONTROLL	3.08	1.26	NO	3.68	4.20	2.60	1.00	1.30	1.30	1.50	167.00	ABNORMAI	MILD	71	15.57	NORMAL	0.47	0.84	0.92	270	ABNORMAL
71	RAMESH	50	M	6	YES	29.29	OBESE	150/90	CONTROLL	3.04	1.19	NO	4.12	5.50	3.20	9.00	1.30	1.10	1.60	213.00	NORMAL	NORMAL	71	16.26	NORMAL	0.86	1.02	0.82	270	ABNORMAL
72	AMUDHA	38	F	2	NO	22.50	NORMAL	120/80	ONTROLLE	2.75	1.19	NO	2.80	4.40	3.10	0.90	1.10	1.90	1.30	128.00	NORMAL	NORMAL	56	15.18	NORMAL	0.91	0.82	1.11	180	NORMAL
73	MAARISAMY	38	M	2	NO	29.07	OBESE	150/80	CONTROLL	2.92	1.01	NO	2.50	4.20	2.60	0.90	1.50	1.20	1.70	147.00	NORMAL	NORMAL	67	17.33	NORMAL	0.88	0.73	1.20	216	NORMAL
74	SHANKAR	47	M	0.1	YES	21.15	NORMAL	170/96	CONTROLL	3.05	1.09	NO	3.88	4.70	2.80	0.90	1.40	1.00	1.50	279.00	ABNORMAI	MODERATE	73	13.18	NORMAL	0.86	0.73	1.18	174	NORMAL
75	VIJAYA	50	F	0.6	NO	24.12	VERWEIGH	190/100	CONTROLL	3.42	1.50	NO	3.74	4.70	3.40	1.10	1.30	1.10	1.40	187.00	ABNORMAI	MODERATE	60	14.65	NORMAL	1.01	0.75	1.36	222	NORMAL
76	ESWARI	60	F	0.1	NO	26.48	OBESE	160/100	CONTROLL	2.32	0.99	NO	3.18	5.80	4.20	1.00	1.20	0.80	1.30	203.00	ABNORMAI	MODERATE	60	15.42	NORMAL	0.84	0.94	0.80	288	ABNORMAL
77	MAARIMUTHU	56	M	2	YES	24.61	VERWEIGH	150/100	CONTROLL	2.89	1.06	NO	3.34	5.10	3.90	1.10	1.40	0.80	1.40	176.00	NORMAL	NORMAL	64	12.98	NORMAL	0.74	0.95	0.78	270	ABNORMAL
78	KABALI	62	M	3	YES	24.80	VERWEIGH	136/98	CONTROLL	3.04	1.08	NO	4.12	5.00	3.80	1.10	1.40	0.90	1.40	182.00	NORMAL	NORMAL	60	13.00	NORMAL	0.80	0.84	0.95	264	ABNORMAL
79	THANGARAJ	64	M	12	NO	21.87	NORMAL	150/100	CONTROLL	3.11	1.21	NO	3.10	5.10	2.80	1.50	2.10	1.50	1.70	332.00	ABNORMAI	SEVERE	69	15.91	NORMAL	1.25	1.33	0.64	290	ABNORMAL

80	PRAKASH	56	M	1	NO	34.57	OBESE	156/88	CONTROLL	3.58	1.33	NO	4.42	5.30	3.00	1.10	1.60	1.20	1.60	242.00	ABNORMAL	MILD	73	17.66	NORMAL	0.89	0.97	0.82	270	ABNORMAL
81	MAHENDRAN	41	M	3	NO	22.22	NORMAL	120/80	ONTROLLE	2.90	1.29	NO	3.14	5.06	3.29	0.98	1.23	0.98	1.28	194.00	NORMAL	NORMAL	60	15.85	NORMAL	0.88	0.59	1.48	186	NORMAL
82	SUNDARAM	49	M	1	YES	28.05	OBESE	120/80	ONTROLLE	3.30	1.24	NO	2.70	4.50	2.70	1.20	1.80	1.00	1.30	175.00	NORMAL	NORMAL	71	15.29	NORMAL	0.78	1.09	0.72	264	ABNORMAL
83	SARANYA	43	F	12	NO	27.83	OBESE	120/80	ONTROLLE	3.27	1.38	NO	3.75	4.50	3.10	1.70	2.20	1.30	1.60	276.00	ABNORMAL	SEVERE	58	18.77	NORMAL	0.97	0.81	2.20	114	ABNORMAL
84	MAHALAKSHMI	31	F	0.3	NO	22.22	NORMAL	130/80	ONTROLLE	2.37	1.05	NO	2.50	4.20	2.50	0.80	1.00	0.90	1.30	110.00	NORMAL	NORMAL	73	12.21	NORMAL	0.80	0.73	1.10	96	NORMAL
85	RADHAKRISHNAN	63	M	14	NO	34.29	OBESE	140/90	CONTROLL	3.30	1.26	NO	2.80	5.30	3.60	1.10	1.70	1.10	1.40	228.00	ABNORMAL	MILD	61	19.50	NORMAL	0.60	1.06	0.57	292	ABNORMAL
86	PANKAJAM	56	F	2	NO	25.91	OBESE	150/90	CONTROLL	2.75	1.27	NO	2.96	4.50	3.00	1.10	1.40	0.90	1.10	153.00	NORMAL	NORMAL	61	16.70	NORMAL	0.90	0.82	1.10	120	NORMAL
87	MANGAYAKANNI	45	F	1	NO	20.23	NORMAL	140/90	CONTROLL	3.54	1.49	NO	2.60	5.10	2.90	1.20	1.60	1.10	1.40	227.00	ABNORMAL	SEVERE	73	16.30	NORMAL	0.60	0.84	0.81	272	ABNORMAL
88	GABRIEL	37	M	4	NO	23.79	VERWEIGH	180/98	CONTROLL	3.00	1.11	NO	3.20	4.60	3.00	1.00	1.40	1.00	1.40	159.00	NORMAL	NORMAL	70	17.00	NORMAL	0.85	0.78	1.10	78	NORMAL
89	MANGALADEVI	55	F	8	NO	29.21	OBESE	180/100	CONTROLL	3.00	1.37	NO	3.65	4.50	2.70	1.10	1.40	1.00	1.30	164.00	ABNORMAL	MILD	69	15.17	NORMAL	0.47	0.93	0.54	294	ABNORMAL
90	KARUNAKARAN	38	F	2	NO	20.93	NORMAL	154/90	CONTROLL	2.84	1.21	NO	3.00	4.20	2.60	1.30	1.00	1.10	1.50	178.00	ABNORMAL	MILD	70	17.00	NORMAL	0.61	0.75	0.80	272	ABNORMAL
91	SELVI	59	F	0.6	NO	21.91	NORMAL	120/80	ONTROLLE	2.41	1.10	NO	2.49	4.30	2.60	0.80	1.20	1.00	1.50	123.00	NORMAL	NORMAL	70	17.63	NORMAL	1.09	0.92	1.18	180	NORMAL
92	RAMESHBABU	45	M	7	NO	26.57	OBESE	140/90	CONTROLL	2.90	1.03	NO	3.14	5.10	3.30	1.00	1.20	1.00	1.30	188.00	NORMAL	NORMAL	64	16.93	NORMAL	1.01	0.76	1.33	168	NORMAL
93	SAKTHI	60	F	0.1	NO	26.48	OBESE	160/100	CONTROLL	2.32	0.99	NO	3.18	5.80	4.20	1.00	1.20	0.80	1.30	203.00	ABNORMAL	MODERATE	60	15.42	NORMAL	0.84	0.94	0.80	288	ABNORMAL
94	JAMEEL	63	M	12	NO	29.13	OBESE	140/96	CONTROLL	3.31	1.38	NO	2.70	4.90	3.30	1.20	1.60	0.80	1.10	176.00	NORMAL	NORMAL	61	17.70	NORMAL	0.71	0.91	0.78	290	ABNORMAL
95	VENKATESAN	55	M	1	NO	23.22	VERWEIGH	140/90	CONTROLL	2.78	1.01	NO	3.30	4.10	2.90	1.10	1.20	0.90	1.20	132.00	NORMAL	NORMAL	61	11.26	NORMAL	0.71	0.92	0.78	256	ABNORMAL
96	RAM KUMAR	46	M	2	YES	26.77	OBESE	158/92	CONTROLL	3.33	1.24	NO	3.60	4.80	2.70	1.10	1.60	1.10	1.40	194.00	NORMAL	NORMAL	76	15.40	NORMAL	0.76	0.64	1.20	72	NORMAL
97	DEVI	40	F	1	NO	25.55	OBESE	140/84	CONTROLL	3.10	1.50	NO	2.90	4.60	2.60	0.80	1.50	0.80	1.10	118.00	NORMAL	NORMAL	73	17.00	NORMAL	0.90	0.97	0.79	264	ABNORMAL
98	SELVAKUMAR	46	M	2	NO	27.51	OBESE	130/100	CONTROLL	3.30	1.23	NO	3.60	4.80	2.70	1.10	1.60	1.10	1.30	260.00	ABNORMAL	MODERATE	60	15.40	NORMAL	0.76	0.64	1.20	0.72	NORMAL
99	RUKMANI	43	F	5	NO	29.77	OBESE	130/80	ONTROLLE	3.41	1.42	NO	3.00	5.00	3.10	1.10	1.50	0.90	1.20	170.00	ABNORMAL	MILD	68	18.00	NORMAL	1.18	0.80	1.47	138	NORMAL
100	RAJASEKAR	55	M	5	NO	23.53	VERWEIGH	150/112	CONTROLL	3.25	1.12	NO	3.48	4.60	3.50	1.10	0.83	0.90	1.30	159.00	NORMAL	NORMAL	70	15.34	NORMAL	0.96	0.71	1.40	144	NORMAL